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# LECTURES

ON

# PATHOLOGICAL ANATOMY.

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# LECTURES

ON

# PATHOLOGICAL ANATOMY.

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*THIRD EDITION,*

THOROUGHLY REVISED.

BY

SAMUEL WILKS, M.D., LLD., F.R.S.

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# PREFACE TO THE THIRD EDITION

THE Second Edition of my Lectures on Pathological Anatomy having been exhausted shortly before the death of my colleague Dr. Moxon, I sought for someone who should fill the place of that distinguished and lamented Physician in the continuation of the work. Some of my hospital friends, however, whose advice I asked, urged me to take the preparation of a Third Edition into my own hands. The reason for this advice was that a younger man, whether from my own school or elsewhere, while in possession of considerable histological knowledge, might introduce this at the expense of the more general descriptive nature of the book, which I had striven to make, as it has always, I believe, been regarded, its distinguishing and most valuable characteristic. It was said that by editing the Lectures myself their essential features would be retained; and as to my fitness for the task, it was pointed out to me that, based as the work was and is upon an unrivalled experience of many years in the post-mortem room of Guy's Hospital, it could never become out of date, while my own interest in Pathology had in no way abated, as is evidenced by my still frequent presence in the post-mortem-room, and by my attendance at the Pathological Society. Being myself also jealous of any alteration in the character of the work I decided to undertake this, the Third Edition of the Lectures on Pathology.

The book will be found of the same size as the last; for although I have made numerous additions, I have omitted many parts which appeared to be unnecessary repetitions, and have entirely re-written some of the chapters.

For suggestions as to these alterations I have been much indebted to Dr. Goodhart. Having a thorough knowledge of the contents of the book and a familiarity, like his predecessors, with the post-mortem-room derived from many years of work, he has been enabled to point out the parts which were obsolete and those which required rewriting.

To Dr. Perry my best thanks are due for reading the sheets as they passed through the press. I hope if the profession is as ready as heretofore to relieve me of this edition, he will have the opportunity of taking a fresh one into his own hands.

SAMUEL WILKS.

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# PATHOLOGICAL ANATOMY

## DISEASES OF BONE

**Malformation.**—*Excess of development.*—Parts that are repeated in series in the body, such as the vertebræ and the fingers, are occasionally produced in more than their proper number. Examples of this are seen in *supernumerary vertebræ;* for instance, we may have thirteen dorsal, each with a pair of ribs to it, or six lumbar. We need scarcely say that along with each additional vertebra all the soft parts which are connected with an ordinary vertebra are present, so that there is rather an additional zone to the body than a mere excess of bone development. The same remark will apply more or less obviously to the other bone malformations.

When an additional vertebra is present in the lumbar region, it is, in some cases, difficult to say whether the extra vertebra is a lumbar, or an additional sacral, piece. Thus, in one specimen, when looked at in front, it appears as if an extra piece had been added to the upper part of the sacrum, but from behind the arches and spine are seen to be distinct, like those of the lumbar vertebræ. Sometimes when there are but five lumbars, the fifth may coalesce by one or both of its sides with the sacrum below.

*Supernumerary ribs* are short processes seen in the neck and loins, and called cervical and lumbar ribs. We have a specimen of *episternal bones*, where two small tubercles are seen growing on the upper edge of the sternum; they are thought to correspond to the coracoid bones of birds. Also a humerus, exhibiting a *supra-condyloid process*, which formerly was catalogued as an exostosis. This process, which is situated about two inches above the inner condyle, forms a hook, and, when a ligament extends from its extremity to the condyle below, a hole or ring is produced corresponding to that supra-condyloid foramen met with in the humerus of the carnivora and some other classes of animals, which serves to transmit the median nerve and brachial artery. Many specimens of *supernumerary fingers and toes* may be found on our shelves. These do not always contain bone, and when bone is present it is often only an excrescence from the bone of the next finger or toe. This multiplication affects chiefly the terminal phalangeal bones, the metacarpus and carpus, &c., being less liable to be implicated, though sometimes the cuneiform bones, &c., may alone be found in too great number.

*Deficiency in development.*—A remarkable form of this is seen in the *anencephalous* fœtuses, of which we have numerous examples, and in which, while the brain is absent, the bones of the cranium are scarcely developed. There may be a deficiency limited to parts of the skull, as in one preparation, exhibiting the want of a portion of parietal bone; sometimes such deficiencies are large, but the most interesting cases of the kind are exemplified in another specimen, where there is an opening in the occipital bone, through which a portion of brain or of its membranes protrudes, constituting an *encephalocele.* These are not always occipital, they may be frontal, and occasionally are lateral; they do not usually pass through sutures. In some rare cases such protrusions come through the base of the skull, and may project into and from the mouth in very strange forms.

A still more common deficiency is a want of union of the posterior arches of the vertebræ, constituting *spina bifida.* Occasionally the bodies of the vertebra may be split or double. The sternum may be cleft, and so may the hard palate, through a failure of union in the anterior arches, a failure which corresponds to the non-union of the posterior arches in spina bifida. The failure of union of the posterior arches may affect the whole cranio-vertebral system, the arch of the skull with the brain being absent, as well as the arches of the spinal vertebræ. In other examples hydrocephalus coexists with the spina bifida. The non-union varies in its extent in the spinal column; some cases show an open canal the whole length of the spine, while others exhibit a deficiency limited to a particular locality. This is generally in the loins, where we find the posterior arches of two or three vertebræ wanting, and the membranes protruding in the form of a sac. This sac is generally about as large as an orange, and usually consists of the spinal membranes filled with fluid, protruding outwards through the opening in the vertebral canal; its inner surface is then lined by the visceral arachnoid, and, therefore, the fluid within it is the sub-arachnoid fluid, and communicates directly with the fluids in the ventricles of the brain. This visceral arachnoid is closely adherent outside to the parietal arachnoid and dura mater, and the latter in their turn to the integument, so that it is difficult to separate these structures, which form the wall of the sac. The medulla or cauda equina, at the spot where the bones are deficient, may continue its way downwards as usual; or if the opening be large the nerves may pass into the sac, and be distributed upon its walls. The nerves go regularly from their points of origin on the cord; some run a short distance on the outer wall of the sac, curve round, and then go through its midst to their exit at the fore border by the intervertebral foramina; others make longer windings on the outer wall before they take their forward course, in which they sometimes stretch back to the cord; on reaching the intervertebral foramina they pass through the dura mater in two rows, and make their ganglia as usual. Spina bifida is not always of this simpler form, which is termed, from its components, *hydromeningocele.* In another form, which is not altogether rare, the central canal of the cord is enormously distended with fluid, and the cord itself is thus expanded into the tumour, constituting *hydromyelocele.* These conditions are analogous to common hydrocele in being irritative dropsies; they probably depend on irritation of the nervous centres in fœtal life. A funnel-like depression or umbilicus in the middle line of the tumour will generally signify the point of insertion of the spinal cord on the wall of the sac. In a case which was tapped the sac contained several ounces of fluid. This was slightly alkaline, faintly opalescent by heat, and had a sp. gr. of 1008.

A simple *want of union,* without protrusion of the spinal contents, is sometimes met with, as in a specimen of atlas, where the two halves are perfect, but are not united, though probably in the fresh subject they were joined by ligamentous tissue. Sometimes one half of the arch of a vertebra is not developed, and thus the spine bends towards that side and a distortion results. One specimen is a very interesting and remarkable example of such an occurrence. We see that the spine is distorted from a fusion of three dorsal vertebræ, arising apparently from this want of regular development; and in the neck we also see that the arch of one vertebra is not completed, but that one half is ankylosed to the arch of the vertebra above, while the other half remains free, and alone gives origin to the spinous process. The spinous process, too, may be sometimes *bifid.* Mr. Lane has shown in his various writings that many of these deformities may arise from pressure due to the effects of laborious occupations. Absence of certain bones is met with in cases of deficiency in the number of the ribs; or in cases where the hands and feet are articulated to the scapula and pelvis respectively; or in cases of imperfectly formed or absent fingers and toes. Such deformities are sometimes hereditary. The man from whom our models of the hand with deficient fingers were taken stated that the malformation had occurred in some members of his family for five generations, but not always in a direct line of descent. Among other abnormalities might be mentioned bifid ensiform cartilage, which sometimes produces a foramen in the end of the bone, bifid ribs, bifid odontoid process of axis. *Ossa triquetra* will be hereafter mentioned. When a complete limb is wanting, it is often due to accident, and is called intra-uterine amputation.

*Irregular development of skull.*—Less striking, but more really important than these obvious variations of bony development (which, as we have said, go with an equal extent of malformation in the corresponding soft parts), are certain irregularities in the normal proportion of development in the several bones of the skull. In normal skulls the several bones bear a nearly constant proportion in size to each other, but sometimes one or more of the bones of the base is too small, and Virchow has shown that the surface growth of the several bones is dependent on the persistence of the sutures, too early a coalescence of a suture checking the growth of bone that should occur along it. This *premature synostosis* may be limited to a single suture, while growth occurs at the others; in this manner the shape of the skull, and with it the development of the facial bones, may acquire characteristic modifications. Synostosis of the sagittal suture stops the widening of the skull, and meanwhile it may grow disproportionately long, or *scapho-cephalic;* synostosis of the coronal suture checks the lengthening, and allows the widening, thereby producing a *platy-cephalic* skull; other imperfections in cranial form will readily be traced from other seats of the synostosis. It is a most interesting question whether these changes in the bone are primary, and give rise to those imperfections or irregularities of the brain which must conform to them; thus a premature synostosis of all the sutures necessarily creates a small head or *microcephalus,* and this is found often enclosing the small brain of an idiot. It is thought by some that this closed smallness of skull causes the smallness of brain by preventing its development. Others have shown that microcephalic skulls are not always synosteal. Thus, Dr. Down, from observations of the skulls of two hundred idiots, concludes that the deviations of the cranium have been rather the sequence of circumstances arresting the development and growth of the brain, and not the result of premature ossification. Extreme microcephalus is found without any ossification of cranial sutures. In the skull of a *cretin* described by His the base was short, and yet its bones were so far from being synosteal that they came apart on maceration, though the subject was fifty-eight years old. The whole skeleton, as usual in cretins, presented puerile characters. Whether in natural development the bone moulds the soft parts, or the soft parts the bone, is as yet undecided, but abnormal changes in either no doubt influence the other. Instances of this we shall presently see.

**Atrophy.**—The simplest condition is that arising from *disuse,* a good example of which is seen in a skeleton in our Museum of a man who, being paralysed in his lower extremities, used his arms for progression; the bones of the leg are in consequence much wasted. After any *fracture* of a bone, which produces injury to its nutrient artery, a partial wasting of the fractured ends may take place. Violent *separation of an epiphysis,* which is a form of fracture far from uncommon in early life, often, though not necessarily, leads to deficiency of development of the fractured bone. The bones, in cases of *congenital paralysis,* share the general smallness of the affected limb—a condition, however, which is rather a result of undergrowth than atrophy. The bones waste in *old age,* the rind of the bone becomes thinner, brittle, and porous, and the medullary canal enlarged and filled with a fatty matter. The effects of age are also seen on the neck of the thigh bone in persons of advanced life, in whom the neck sinks to a more advanced angle with the shaft; it is also shortened, and of open cancellous structure; its condition requiring care in distinguishing it from the results of fracture. A somewhat similar atrophy to that often seen in old persons is met with in limbs which have long been inactive from disease of the joints, or from the patient being bedridden; the bone is found to have a thin shell, and the medullary canal is filled with a soft yellow matter, consisting almost entirely of fat; the cancellous structure of the bone is also occupied by it. Atrophy may also arise from *pressure*. This is especially seen in aneurysm of the aorta, where the tumour, by constant pressure against the spine, gradually erodes the bones until the bodies of the vertebræ are destroyed, and in some severe cases the canal laid open, the intervertebral cartilages remaining comparatively unaffected. The loss of substance is a simple destruction of the bone, and in nowise can be called ulceration or caries. In the same way the sternum, ribs, &c., may be affected, as may the long bones from the immediate pressure of aneurysms. Sometimes the bone appears lifted and expanded before the advancing aneurysm. Thus, parts of the sternum and clavicle may rise like an incomplete shell before an aneurysmal tumour, so, perhaps, making it difficult to distinguish the aneurysm from a pulsatile growth expanding the bone. It seems improbable that the bony matter thus apparently lifted is really the natural bone, for this is not extensible and ductile; we must suppose that, the original bone being removed, a growth of bone is produced in the periosteum, as this is thrust before the advancing tumour into new positions and shapes. In the calvaria we may often notice deep depressions along the sides of the longitudinal sinus, produced by the pressure of the *Pacchionian bodies*, and *tumours* of all kinds may produce like effects on the bones.

Apart from these manifest causes, we may often find thinning of the bones; thus in the cranium, and especially in maniacs, this condition may be seen, although in some of these the skull may be very thick, and in others very thin, or it may be thick in places and thin in places. Sometimes on holding up a calvaria to the light, we may see it almost as thin as paper on each side of the median line; and sometimes there is a depression on each side, and a raised ridge of bone along the temples. In a skeleton, which is said to be affected with *rickets*, the cranium is seen to be remarkably thin. This condition has of late been considered to be associated with congenital syphilis, as we shall presently mention. Dr. Hale White, who has studied the question in adults, believes that the atrophy of the bones is due to pressure, for he has generally found it in cases of tumour of the brain, and especially where effusion has also been present. The bones at the base of the skull, too, have also been involved. It must be remarked, however, that it is not uncommon to find the latter atrophied where there has been no evidence of pressure, nor indeed of any cerebral disease; for example, the petrous bones, orbital plates, and adjacent parts of the base, may have become quite diaphanous, and the point of the scalpel be easily made to penetrate them. It is a condition accidentally met with, and does not indicate, as far as we are aware, any distinct pathological state.

The thinness of the orbital plates must be remembered in cases of penetrating injuries about the eye, for occasionally these plates are broken by slight causes, such as a thrust with a tobacco pipe, and this when there is no external sign of the severity of the injury; perhaps, too, for a time there may be no symptoms, even when the brain is subsequently found to have been penetrated.

**Hypertrophy.**—*Relation to inflammation.*—Low augmentative processes are registered in the bones more obviously and enduringly than in any other component parts of the body, because of the permanency of the bone produced. Very slow augmentative processes of low intensity produce increase in the size of the bone without giving rise to the heat, pain, &c., which are the clinical signs of inflammation. These processes are called by the general name *Hypertrophy.* When the cause of production is somewhat more intense, or acts with greater rapidity, the vascular disturbances of inflammation attend the hypertrophy; and when the intensity is yet greater, the bone ulcerates, softens, or perishes in larger measure by necrosis. Considering this series of changes only anatomically, so that the heat, pain, &c., are out of recognition, it will be clear that hypertrophy, the simple, slow augmentation, cannot be *anatomically* distinguished from the milder inflammation which is characterised by mere vascular disturbance without destruction of the bone, and with new formation upon or in it. But the higher inflammation, resulting in ulceration, caries, or necrosis, stands plainly distinguished from hypertrophy, and is called by the name *inflammation.* It is necessary to clearly understand this relation of the word inflammation to the anatomical facts we are considering. Inflammation is the name of the process of which hypertrophy, caries, and necrosis are the anatomical results; but the process which results in hypertrophy is often latent and unrevealed by symptoms, so that hypertrophy then exists without evident inflammation. On the other hand, caries and necrosis are always accompanied by the clinical phenomena of inflammation, so that they are commonly and conveniently called inflammatory. It is very important to understand the sense in which the word is applicable. The continuity in nature of the low and high degrees of inflammation, which we have just been speaking of, is fully and clearly proved in the progress of severe inflammation of bone; for we see the destructive effects in the focus of the severe inflammation encircled by productive effects of the milder action around. The inflammatory action decreases with the distance from its focus. In a femur, for instance, the lower third of its shaft may be partly necrosed, the dead piece representing the focus of the inflammation, but around it new bone has formed; and this formation is less in quantity and more normal in quality as you proceed away from the dead piece, until the entirely healthy bone at the upper part of the shaft is reached.

The indestructibility of bone by common decay enables us to prepare specimens which beautifully display the destructive and constructive areas of inflammation. The constant production of bone around the seats of grave inflammation makes obvious to us the fact that the increased blood supply which accompanies inflammation will cause bone to undergo hypertrophy. Such hypertrophy is, however, not anything proper to inflammation; we often see zones of pure bone in a similar way surrounding cancer growths, especially in the cranium; indeed, any cause of active hyperæmia will produce hypertrophy of bone, as, for instance, when under ulcers of the skin a lump will arise on the tibia, &c.

We understand, then, that when thickenings such as those surrounding an inflammation occur without any inflammation being present to explain them—we mean without the heat and pain of inflammation, so that the augmentation of the bone is the only thing recognisable—we speak of it as a primary thing, with a nature of its own, and call it *hypertrophy;* of course knowing all the while that some cause of irritation, equivalent to the inflammatory fluxion, did in reality act to create the hypertrophy when it was produced.

In inquiring for the inflammatory symptoms we must remember that these new formations of bone persist long, perhaps for ever, after they are once made, so that the action that created them may easily be out of mind when the hypertrophy is met long years after. In this wide sense hypertrophy has a most extensive application, and includes a great number of morbid changes which arise from distinct causes, show diversified courses, and, in short, are brought together only by their anatomical resemblances.

*Periosteal and endosteal hypertrophy.*—Now, if we take a series of such examples of the states surrounding inflammation, we shall find by close inspection that the bone increases in two ways; thus: first, some new bone is added to the surface of the old; and, second, other bone appears filling the natural medullary spaces, so as to make it more solid and denser. We may here, then, usefully remark a fact more easy to realise than to explain; namely, that some cases of the spontaneous or non-inflammatory increase of bone, which is called by the general name of hypertrophy, offer us an actual enlargement of the size of the bone without increase of its density; while others, on the contrary, show no superficial enlargement, or but little, while the density and closeness of the texture of the bone is increased through the filling up of Haversian spaces, until the bone resembles ivory. Now, most cases of hypertrophy of bone include both these kinds of increase—the superficial and the interstitial. When the increase is in density only the name *sclerosis* was given by Lobstein. The ultimate condition of the bone is, no doubt, much the same in all hypertrophies, whether they arise from inflammation or not; and thus in a piece of femur (which may be in reality from a case of ostitis) the structure is very dense and like ivory, as it is also in another slice of an hypertrophied cranium, which is probably not inflammatory. It might be thought that a bone presenting the ivory-like condition must be very different from an ordinary one; but, although it may be so to outward appearance, the microscope does not show much except to a practised eye. The greater compactness of *old* bone arises from a filling up of the spaces in the bone with concentric bony laminæ.

But formations of bone sometimes arise on the surface by ossification of the periosteum. The method of this difference lies here, that, when the bone enlarges on its surface, the new bone is produced directly *from the periosteum;* whereas, when the bone condenses without enlarging, the new bone is formed *from the medulla* lining the cancelli.

In either case the bone formed has the general characters of osseous tissue (*tela ossea*), with which we are familiar, and it passes through its proper developmental stages. Thus, when young, the new bone on the surfaces of the old is spongy, being formed of spicules mutually uniting to make a meshwork, a primary cancellus, in which the unossified remainder of periosteal tissue is seen supplied in each mesh by a vessel. Then the ossification of this periosteal tissue proceeds until the vessel is surrounded by close bone. After some time the formation of medullary spaces will occur in the deep part of this new dense bone, and so a cancellous structure again returns. But this secondary cancellation is not the same as the primary, for its meshes are occupied by marrow and not by unossified periosteum as the first meshes were. So we see that fresh-formed bone is less organised, and is wanting in adaptation of its parts to the situations they are to occupy, the surface of the new bone and its deeper portions being much alike; but, as in all other permanent formations, in process of time the new part tends to assume the characters of structure and arrangement proper to the healthy tissue, and so the surface becomes dense and the deeper part cancellous. It is thus that outgrowths of bone acquire even marrow canals, and in like manner the marrow canal is modelled out through the callus of badly united fractures. The knowledge of this will enable us often to infer approximately the age of a bony hypertrophy, and to understand why some adventitious bone is close-textured, being new, while other is cancellated, being old. Notwithstanding this, we cannot always explain the differences of solidity in new bone as due to the stages of development. Indeed, the ivory-like "eburnation" of some new growths attains to a degree of density much greater than that of the ordinary close-textured, newly formed bone. This is especially true of the ivory exostoses of the cranium and orbits.

*General and local hypertrophy; varieties of hypertrophy.*—So different are the appearances and the clinical nature of these bony overgrowths that it is difficult to distribute them into varieties for description. They are usually divided into *general and local* hypertrophies, though the distinction is not very definite. Still viewing at large the cases of hypertrophy, we see that, in some, the change affects many bones, or even all the skeleton, while in others the increase is limited to one bone, or even to the small part of a bone.

Let us first consider the more *general* forms of hypertrophy—we mean the more generally diffused overgrowths of bone. A convenient term for these is *Hyperostosis.* It will correspond with the old and accepted use of this term, if we mean by it those enlargements of bone that are less circumscribed upon the bones they affect, generally implicating the whole bone, or even many bones. More circumscribed formations of bone are called *Periostoses*, and if these are so defined as to resemble tumours, they are called *Exostoses.*

The simplest form of general enlargement, or *hyperostosis*, arises from increased function, as seen in the case of that same skeleton of the paraplegic man who used his arms for progression, which we mentioned just now. It is perhaps hardly fair to call this hypertrophy ; it rather is the state of bone corresponding to thorough muscular development, and can scarcely be construed into morbid anatomy. Quite different is the case with some specimens derived from the skeleton of a man sixty years old, whom we attended, and who was affected with general hypertrophy of the bones. In these it may be observed that the surface is porous and has a singular mortar-like look, being whiter than natural, and not having the usual surface-structure of bone. Dr. Goodhart, who made the inspection, says, "When fresh the periosteum appeared natural, but the bone was pink throughout, and though firm and heavy, much softer than normal bone." "Those bones that could be examined, as the cranium, ribs, pelvis, thigh bones, &c., were affected, but apparently not the bones of the face. The left clavicle, for instance, was two and a half inches in circumference, and on section showed a uniform surface, with hardly any medullary cavity, and no cancellous tissue. Section of the femur showed two parts of the bone distinct from each other, one being the old compact bone immediately around the medullary canal, and external to this a good thickness of more vascular new bone." The femur weighed 2 lbs. 14 oz., and was from six inches and a half to eight inches and a half in circumference. The pelvis was of the form usual in rickets. The microscope showed that the diseased bone had undergone a considerable change ; the Haversian canals were enlarged and of irregular shape. During life the bones were observed to enlarge for twelve years, and the man died of fixation of the chest through disease of the ribs. Since this description was given, Sir James Paget has written an elaborate paper on the subject in the 'Med.-Chir. Transactions,' and has styled the disease *osteitis deformans.* His patients, mostly men, had grown shorter, whilst the bones had become bent and enlarged. There had evidently been a chronic inflammatory change whereby the bone had become rarefied and softened, and so given way. This change was near the medullary cavity, for on the surface the bone was more dense, or sclerosed. There is, therefore, in these cases a curious combination of condensation and hardening, with softening and rarefaction. The cranium also is usually thick and soft.

In the first case mentioned, a new growth of a sarcomatous kind was found in the skull, and Dr. Goodhart has remarked a similar fact in other cases of hypertrophy of the bones; whence he concludes that the prolonged inflammatory process may at last put on a malignant character. And it is certainly a remarkable fact that in most of the hitherto recorded cases of osteitis deformans malignant tumours of one kind or another have been found at the autopsy.

Progressive enlargement of the skeleton affecting some parts more than others, and this so as to cause tumour-like masses in places, has occurred in several recorded instances. In Dupuytren's case the growths began in a sucking child at many points in the trunk and extremities, and continued enlarging until death. The adjacent ends of the femur and tibia grew to the size of "knobby potatoes," while other bones were less affected, and others, natural. In Saucerotte's case a man, thirty-nine years old, grew in four years 59 lbs. heavier, while his flesh wasted away, his head grew too large for ordinary hats, and his eyes stood out through pressure to a level with his forehead.

Whether these cases are all of the same nature, and if so, what is that nature, are questions we must yet hold doubtful; it is natural to ask after an association with syphilis or rickets as causes, for there is no doubt that either of these disorders will give rise to enlargement of bone. There is, however, no sufficient answer to these questions. Syphilis has generally not been present; but in some cases of general enlargement of bones, some of the bones, say the tibiæ, are found curved as in *rickets.* The porous form of hyperostosis has been called *osteoporosis.* We have many examples of it without any history. In one case all the bones of the skeleton are much enlarged; we see that the sections display a cancellous structure, and the bone, instead of being compact, has the appearance of mortar. This is especially well seen in the skull. The grooves for the meningeal arteries are very deep. Now, the tibiæ have rickety bends, so as to suggest that such a disease may have given rise to the change.

Remarkable examples of hypertrophy are frequently met with in the removal of the calvaria, where the bone is often found twice its usual thickness: the increase may be uniform, or greater in some parts than others. This thickening of the cranium has also been ascribed by some to rickets; while others, with Virchow, ascribe this view to a total misconception of the nature of rickets. We will speak subsequently of this question. If we had any knowledge of the nature of that general progressive porous enlargement of the bone or osteoporosis, of which we have given cases, we might probably find these thickenings of the skull to be of the same nature, but less extensive. The peculiar porous appearance of the bone is generally present in them; but, unfortunately, there is no history in connection with the cases.

Another very terrible form of enlargement of bone is a hyperostosis, generally limited to the bones of the skull and face, increasing them enormously, so that in Fourcade's case, figured by Lebert, the macerated skull weighed 8¼ lbs., the lower jaw 3 lbs. 6 oz., the whole, and especially the facial aspect of the skull, being horribly deformed with crowded and heaped and mostly rounded outgrowths, very hard, and not at all resembling the porous condition just described.

From the lion-like look and the real likeness of the disease of the bone to the hypertrophy of the soft parts of the face, which constitutes the kind of leprosy called "Leontiasis," the name *Leontiasis ossea* has been given to this disease. It has been found associated with a similar condition extending to other bones of the skeleton, but, on the contrary, we more frequently meet such an overgrowth limited to one or two bones of the skull or face, or to parts of a bone. Thus, the sphenoid or malar, for instance, may grow into tumours; or more frequently the jawbones swell generally, or at a circumscribed part, producing enlargements which, however, partake more of the nature of tumours. Some of these, implicating for the most part the walls of the frontal sinuses or of the antrum of Highmore, are found exceedingly hard like ivory, and are known as *ivory exostoses.*

Under the name of *Osteophyte* one would widely include every sort of new growth of bone, whether hypertrophic or inflammatory.

This term, indeed, is used generically, and would include the kinds of general hypertrophy we have hitherto dealt with, but it commonly is applied in a more limited signification to name *circumscribed additions to the surfaces of bones.* It was believed by Lobstein that these superficial formations or osteophytes represented the ossified blastema from various sources, which their forms would indicate. The diffused osteophyte coming from under the periosteum, the retiform from periosteum itself, the styliform from the tendons and aponeuroses, and the cauliflower-like from the interstitial tissue.

These names are not now much used, but if we look at many of the bones in our Museum plenty of examples may be seen to which they would be applicable. Thus we may mention a very common affection of the spine, in which new bony matter is found on the bodies of the vertebræ, uniting them together. More or less of this condition is seen in a very large number of the bodies we daily examine, especially in those of hard-worked men. Some have called this condition *ankylosis* of the vertebræ, or ossification of the ligaments, but what we find in the first instance is an ossification of the surface of the intervertebral substance between several of the bones on one side, whereby slight bony projections are produced, the ossification not extending deeply into the fibro-cartilage. At a further stage these several exostoses may unite on the bodies of the vertebræ, and thus a mass of bone is seen lying on their surface resembling a thin lath of osseous material fixed upon the spine; or, sometimes, as if a quantity of soft bone (as it were mortar) had been poured upon it and then hardened. We often find this on one side alone; and if the spine be curved, it is on the side of the concavity.

Mr. Aston Key drew attention many years ago to these bony growths on the spine as a possible cause of paraplegia by their pressure on the medulla. His specimens are now in the Museum, and his paper on the subject is to be read in the third volume of the 'Guy's Hosp. Rep.' There is no proof, however, that these outgrowths are competent to produce such a result; but cases have been recorded where exostoses have protruded into the spinal canal, and pressed on the cord. In connection with this subject we may remark that Mr. Bland Sutton has long studied the changes which the bones undergo in caged animals, and has observed the remarkable softening, or rickety condition, which takes place more especially in monkeys and lions. He found the softened vertebræ bulging into the spinal canal, and sometimes around them an outgrowth of bone taking place, which squeezed the cord and produced paraplegia.

There is a variety of osteophyte called by Rokitansky *puerperal osteophyte*, of which we have mounted a specimen as an apparent example. He states that during pregnancy a new deposition of bone takes place on the inner surface of the cranium especially, seen as a slight layer along the sides of the longitudinal sinus, and also in patches on other parts of the calvaria and base. A specimen, from a young woman who died at the fifth month, shows certainly a new layer in the form of slight elevations along the upper surface of the skull and on the frontal bone; but we question very much whether it is related to the puerperal state. There must be much uncertainty of this until more extended observations are made, for such irregularities are constantly met with on the inner surface of the skulls of persons dying from a great variety of diseases.

A case is recorded of bony growth both within and without the skull which augmented at several successive pregnancies, and the puerperal osteophyte is said to occur on the outer surface of the skull and facial bones. Virchow thinks it is like puerperal endocarditis in its relation to the puerperal state. We have no experience of it.

The prominences of bone which surround joints in the state called *chronic rheumatic arthritis* are strictly osteophytes, though the definite disease they arise from removes their consideration to its own place. All superficial inflammatory formations of bone would likewise deserve the title, but where any known cause of inflammation has produced them, their consideration is merged in that of the inflammation itself. If produced by more recent and more acute inflammations, as in caries, these osteophytes are spongy and spicular. In chronic rheumatic arthritis they are rounded and dense.

When an osteophyte forms a bony tumour on a circumscribed part of the surface of a bone, it is called an *exostosis.* This term is given when the growth is of such a prominence as to pass beyond the proportions of a mere bump or elevation. Such elevations as are mere bumps are called *nodes;* and the distinction generally corresponds to a real difference of cause, for nodes are mostly traceable to injuries, or to the neighbourhood of ulcers, or to ossification of old syphilitic periostitic patches; whereas, on the other hand, exostoses are generally of unknown origin, and correspond more to the nature of tumours to which their surgical treatment practically allies them.

The distinction between an exostosis and an ordinary osteophyte is not very defined; speaking generally, any diffused new bone, on and distinct from the old bone, would be an osteophyte; but an outgrowth of the surface, not showing a distinct demarcation, would be an exostosis. Exostoses are of various forms, and may be met with as projecting processes on the long bones, as seen in many specimens in the museum. They, however, are not indifferent in point of situation, but have certain seats of preference. Thus a very frequent position is the end of the great toe; an exostosis often occurs in young people as a round tumour attached to the end of the last phalanx; a section shows that it is developed from cartilage, the latter forming its circumference, while the centre contains the bone or ossified portion. Such tumours afford good opportunity for witnessing the process of ossification from cartilage. Sometimes they occur all over the body, and are called "*multiple exostoses.*"

Exostoses on the skull have often a deep furrow round their base. Exostoses may occur as isolated rounded growths, perhaps of considerable size, on the outer or inner surface of the cranium, and if the latter they may produce important symptoms of brain irritation. They may hold any situation indifferently, but we think attention should be specially directed to those which form on the inner surface of the skull, chiefly on the frontal bone around the crista galli. These are not very uncommon, and are found accidentally as one or two little growths, like small mussel shells. Their number may be greater, and their form less distinct, until the whole interior of the frontal bone is roughened with projections or the same state is found to extend to neighbouring bones, or even to the whole interior of the skull, reminding us of the nodular hypertrophy of the facial bones before mentioned. In these latter severe cases epilepsy or idiocy has accompanied the condition.

The larger growths, isolated and often nodular on the surface, appear to go inwards through the dura mater; but this really extends very thinly over them. They are hard to tell from bony growths *in* the dura mater, which we shall presently speak of. The gathering of the intra-cranial exostoses around the region where the sinuses are late in developing, and where, therefore, unusually protracted activity is manifested, would suggest this greater activity as a cause of these growths; and in support of such a view we have the frequent occurrence of exostoses of the long bones at points corresponding to the epiphysial cartilages, where activity of growth is continued long.

**Inflammation.**—In speaking of hypertrophy of bone we have followed the common idea of the dry hard bone of the skeleton. This simple notion of bone suited very well for the description of the various kinds of augmentation and alteration of its form, which constitute hypertrophy; but it is most important, now that we come to consider the more violently active changes in bone, to remember that the hard bone of the skeleton is only part of entire bone; indeed, *the bone of the skeleton is but the skeleton of the bone.* In a living bone the hard osseous tissue, or *tela ossea* proper, is covered outside with periosteum; the cavities or cancelli within it are filled with medulla; vessels, either arteries or veins, permeate the substance running in the Haversian canals; even the lacunæ are occupied by cells, whose processes stretch in the canaliculi to communicate with the contents of the lacunæ around.

A living bone contains all these soft parts, and its life is maintained by them. Further, it commonly also is furnished with cartilage at its articular extremities, whilst ligaments, tendons, muscles, or fat are more or less intimately united with its surface, altering not only the periosteum but the texture of the true bone at the points of union, so that, if we carefully consider the surface of any bone, say of the femur, we soon learn to distinguish easily those parts which have been in continuity with the several kinds of tissue which enclose it. We see where the fleshy muscle took its rise, where a particular tendon was attached, or where the adipose tissue lay in contact with the bone about the joint; and can discern altogether different kinds of surfaces, which we find then quite characteristic, so that in this way we can tell by examining the bone whether flesh, tendon, or fat, was connected with any given part of its surface. But the most important connection of the hard *tela ossea* is not this anatomical connection with the parts around for the purpose of supply; it is rather the historic or physiological connection which it has with the soft parts, in that the bone arises from the cartilage, periosteum, or medullary tissue, (ultimately nearly all from the medullary tissue,) and it is always ready to return into the soft state again. The dry bones we are familiar with all our life impress us with a natural but false idea that the bone is a permanent and passive sort of thing, having no proper vitality or changeability.

The deepest layer of periosteum close down upon the bone (at least in growing bones if not during the whole of life) present characters closely resembling the tissue of bone itself. The cells of the periosteum have structure and arrangement resembling those of bone lacunæ, and only need impregnation with earthy salts to constitute true bone. In this state of transition the deep periosteum is called *osteoid tissue,* and is, in fact, young bone, for it is by this simple transformation of the deep periosteum into osseous tissue that the bones grow in thickness. A corresponding but reversed activity is normally present in the marrow cavities, for as the bone increases outside, the marrow cavities form in the older inner texture. These cavities arise thus : the calcified tissue loses its lime in the area that is to be a cavity, and the lacunæ-cells, set free from the lime, multiply, producing numerous small cells in their place, many of which closely resemble the cells of lymph or white blood-cells, but they generally have a reddish colour. Some of these cells may take up fat to form the adipose tissue of marrow. A fresh interest attaches to this medullary tissue since the arguments and observations of Naumann, by which he endeavours to prove that the reddish lymphoid cells are really young red blood-cells, and that the red blood is thus formed in the marrow of the bones.

In the multiplication of the lacunæ-cells some of them show the following variety :—The nuclei of these cells multiply within while the cells grow, and are then found very large and containing a number of nuclei. The large multinucleated cells so formed are called myeloid cells, and are very characteristic of developing marrow. We shall presently see their relation to certain bony tumours. We mention all this because it is necessary that the history of bone formation should be borne in mind, or we cannot follow and comprehend the various results of inflammation in bone. By dwelling on the natural history of bone formation we are taught to regard bone as very far different from the mere petrified lever or shield it is naturally at first thought to be.

True it is that, while the vessels and lacunar cells of the bone are encased and fast set in stony substance, the vital processes must be very restricted, but now that we know, first, that each lacuna contains a living cell, and second, that this living cell is capable of so far ruling over the domain around as to determine the absorption of the lime-salts, thus setting itself free from its stony prison, we recognise that the tissue of bone in the matter of inflammatory and other action has only a temporary disadvantage in comparison with other tissues. Its forces are slow to mobilise, so that it may be overwhelmed in a sudden attack of inflammation, because its cells and vessels are not free to exert those powers of accommodation which would make their activity equal to the emergency. But if the inflammation is less severe, so that time is given for the change the bone is to go through, a very interesting process takes place which, better than any other thing, serves to prove the individual activity of bone-cells. If we look at microscopic sections of inflamed and necrosed bone, we see that the outlines of the Haversian canals of the solid and of the trabeculæ of the spongy bone are not straight as they should be, but are made most irregular and eroded-looking, through the hard bony surface being pitted deeply with little pits called *Howship's lacunæ.* These pits are filled in the fresh and living bone with a soft granulation tissue; they often sink deeply into the bony surface. Now, if we compare these pits with the lacunæ we see that each pit is in the position of one of the lacunar territories, so that no doubt will remain that each pit represents the area around the lacunar cell which has had its stony salts dissolved out. The limitation of this solvent action to the figures of the lacunar territories will leave no doubt that it is to the activity of the lacunar cells that the solution is due. Our knowledge of this activity of the lacunar cells is due to Virchow, who has shown how the *tela ossea* actively works in the inflammatory process. The lime-salts are removed from around the bone-cells, forming thus the Howship's lacunæ; and the bone-cells, set free, proceed to multiply, and so produce a quantity of new tissue, which is like ordinary granulation tissue. We think that so much as this is fully proved, and that the granulation tissue thus formed may either perish in the spread of the inflammation, or, under favorable circumstances, repair the disease by forming new bone or fibrous membrane; but we think it is not shown that pus is so formed; it is probable that the progeny of the bone-cells only tends to repair, and not to pus formation. It might naturally be thought, and has been thought by some, that pus can dissolve bone. This idea seems favoured by the spread of abscess in and through bone, as though by liquefaction of the bony tissue. But it has been proved by experiment that pus has no solvent power on bone; and it is agreed that the spread of suppuration in bone is a vital process in which the living cells of the bone are the essential agents. If the bone with its cells die no solution of it occurs, but it must then be cast out as a dead body, or "*sequestrum.*"

The inflammation of bone produces results like those of inflammation in other structures; but the hard inextensible character of the *tela ossea* of course prevents any rapid accommodation of its tissue to very great vascular changes. Having this unyielding nature it is unable to go through intense attacks of inflammation, and consequently such severe attacks very frequently cause death of the portion of bone that is implicated in them. Death of bone is called necrosis, and it is, indeed, a very frequent result of severe inflammation of bone, but otherwise we find the inflammatory process in bone to have degrees and varieties corresponding with the degrees and varieties observed in inflammations of the soft parts. Thus, inflammation of bone is chronic or acute; plastic or suppurative; simply ulcerous; unhealthily ulcerous, then called *carious;* or gangrenous, then called *necrotic.*

The active inflammatory changes take place in the soft parts of the bone, its periosteum, or its medullary membrane. In some cases both these are implicated, in others only the one or the other. When both are involved, and thus the entire bone is included in the scope of the inflammation, it is called by the general name *ostitis;* when the action is limited to the periosteum it is called *periostitis;* when limited to the medullary membrane, *Osteo-myelitis.* This latter affection may follow the extensions of the medulla throughout more or less of the loose cancellous texture of the bone.

It will be seen that under the term inflammation two very different conditions are observable. The one, where the bone is rarefied, and the other, where it is hardened or sclerosed. In the latter case the inflammatory process is seen to begin on the surface in connection with the periosteum, and in the interior, or medullary canal, in connection with the endosteum, until this is filled with osseous substance, and the whole shaft has become dense throughout. The medullary space, and Haversian canals, are filled with a dense earthy material, causing the canals to disappear, and the bone becomes as hard as ivory.

*Periostitis.*—The periosteal membrane may become inflamed from an external cause, as a blow, or from a constitutional cause, as scrofula or syphilis. The membrane becomes highly vascular or hyperæmic, and after a short time an exudation occurs in and beneath it, by which it becomes swollen and soft, and so is easily stripped off the bone ; at a further stage lymph is formed, and this is, for the most part, beneath the membrane, and if circumscribed constitutes a node. This new product may be absorbed and the part return to its natural condition, or it may undergo further development, especially if a large part of the surface is affected, until a diffused layer of it may become bony, and be connected to the shaft by blood-vessels. Thus, an additional layer of bone is added to the old, the periosteum itself being somewhat thickened. The result of this process will be seen upon many of the bones in the Museum. It appears as a distinct new layer added to the circumference of the shaft. A small mass of lymph is, however, generally absorbed. We might naturally inquire whether the inflammatory product, which thus ossifies and adds to the size of the bone, is yielded by the bone or the periosteum ; no doubt the chief part is produced by the periosteum, which is much more rich in vessels than the bone it covers. Experimental transplantation of the periosteum into the soft parts has proved its tendency to form bone ; any piece of periosteum, except pericranium, when it is successfully engrafted in subcutaneous tissue changes to bone, passing often through an intermediate stage more or less perfectly cartilaginous.

Any displaced periosteum in cases of fracture or abscess ossifies, while in cases where portions of the periosteum slough no bone is formed at those parts. This might suggest that ossification is limited to the periosteum, yet it has been proved that the ossification which commences in the periosteum will extend to the tissues around, and that not only to the fibrous tissues, but sometimes even to the muscles. Thus we become aware that periosteum and parts around will certainly give rise to bone. But it is equally certain that the bone itself, *i. e.* the medullary tissue in the cancelli of bone, will produce new bone. This we see in the ossifying granulations which spring from the exposed articular ends of bone in ankylosis of the joints. Such ossification from the medullary tissue is sufficiently evident also in the common process of condensation of the bones, which is nothing else than ossification of the products of the medullary tissue lining the cancelli. Hence we may conclude that the ossific new product in periostitis is yielded chiefly by the periosteum, but partly by the bone. In the case of the pericranium it is chiefly from the bone itself that the new bone comes, for the pericranium is much less vascular than ordinary periosteum, while the bones of the skull are rich in vessels. If the bone be exposed granulations form on the surface; these are composed of a mass of cells which have been called "osteoblasts," and which soon become converted into bone-corpuscles, these subsequently giving off filaments corresponding to the canaliculi.

*Acute suppurative periostitis, periosteal abscess, or acute necrosis.*—When the inflammation is extensive and severe, pus forms under the periosteum, and raises that membrane off the bone to form an abscess around the bone. Thus arises a terrible and very dangerous disease, which is not very uncommon; it nearly always occurs in boys, and is usually ascribed to some injury which is generally not severe, although often this history is altogether absent. The danger that attends it arises from its disposition to set up a very grave form of pyæmia, during which abscesses occur in the heart and kidneys much more often than in pyæmia from any other cause. It is intensely infective, and micrococci are found in the medulla. These are in chains or groups, and called "streptococci." In a case of Mr. Golding-Bird's the "sour, sweaty smell" described by Cheyne as characteristic of micrococcus fermentation, was powerful in the interior of the heart. The disease is not usually limited to the periosteum; indeed, some surgeons maintain that necrosis of the bone does not occur unless the medullary membrane is implicated, and they would place this infective disease under the next section, *osteo-myelitis.* Certainly section of the bone usually reveals an inflamed state within; lymph and pus are found within the medullary cavity and cancelli; yet in many cases this is not so, but the bone is dead, while the interior exhibits no signs of former inflammation. Sometimes the disease kills very rapidly, as, for instance, in six or seven days; indeed, before pus has time to form in large quantity. The periosteum is then found detached and the bone separated from it by a dirty, brownish, turbid fluid, consisting of pus in which some altered blood is mixed. If the patient live longer, say for two or three weeks, a large quantity of pus is found around the bone, the bone itself being quite naked and white, or, more strictly, of the colour of milk of sulphur. The disease generally stops at the epiphyses, but not always; it very rarely, however, reaches the joints. It nearly always attacks one of the long bones, more generally of the lower extremity, but it has been known to affect the pelvic bones, pterygoid process, &c. We have seen it limited to the middle phalanx of one finger in a case rapidly fatal with the characteristic pyæmia. Fortunately, however, the disease is not always of this great severity. It may be only less rapidly fatal, lasting six weeks or six months; but in happier cases no fatal conditions arise. It is then that we see an amount of restorative power as surprising as the terribly rapid, fatal, issue in the grave examples is startling. New bone forms the periosteum around the old, like the bark of a tree around the old wood. The top and bottom of this new case, or capsule of bone, are united with the epiphyses, and when the piece of old bone, or *sequestrum*, is removed, this capsule of new bone gradually contracts to the dimensions of the old, though seldom quite so far, and then assumes the proper figure of the bone, having a medullary canal within it as before. The history of these milder cases, however, here merges in the general history of necrosis, which we shall presently describe. Mr. Tom Smith showed at the Pathological Society a remarkable case of an infant two years old the subject of *hæmorrhagic periostitis*. Blood was found effused under the periosteum of the shafts of the long bones, and also between the epiphysis and the shaft. It was probably a kind of scurvy acting on a rickety bone.

*Osteo-myelitis.*—In speaking of periosteal abscess we mentioned the frequent co-existence of inflammation of the medulla of the bone with the periostitis in that disease. Indeed, the cases are rare in which the periosteum or medulla alone is inflamed. Yet though this is true, we find it convenient to recognise as distinct classes those cases in which the periosteum on the one hand, or the medulla on the other, is the chief seat of the disease. Using the term osteo-myelitis as a general name for all cases of inflammation affecting the marrow canal and the lining of the cancellous tissue, the application of it becomes very wide, and includes affections of many degrees of severity and of several distinct varieties of disposition. Thus, in the neighbourhood of diseased joints the cancellous bone has its medulla nearly always, if not quite always, in a state of inflammation, the result of which inflammation differs according to the disease in the joint. For instance, in chronic rheumatic arthritis the inflammation is generally plastic, and leads to induration of and outgrowths from the bone. The same is the result in chronic traumatic inflammation; but in scrofulous disease of the joint the bone is generally very thin, the effect of the inflammation being an absorption of the *tela ossea* until what is left is often very fragile and greasy, becoming pellucid and bending under pressure of one's fingers. These forms of osteo-myelitis, however, come more properly under the history of joint diseases. Besides these instances of osteo-myelitis, which may be properly regarded as incidental to the course of other affections—though some think that many so-called strumous diseases of joints arise in this way—we meet with cases where the inflammation of the medullary tissue is undoubtedly the chief and primary disease. These cases fall into two groups: the first where the inflammation is diffused in the interior of the bone indefinitely, *diffused osteo-myelitis;* the second where the inflammation seizes upon a limited spot in the bone, *circumscribed osteo-myelitis.*

*Diffused osteo-myelitis* is found both as an *acute* and as a *chronic* disease. Acute diffused osteo-myelitis is a very important malady, and has received much attention since the part it plays in the production of pyæmia has been recognised. It is, indeed, the most frequent cause of pyæmia after amputations and resections, and also after severe blows on the head, such as inflame the cranium and its sinuses without fracture. When the bone thus affected is examined, the medullary cavity and cancellous substance are found to contain pus, which either fills these so as to form an abscess in the cavity, or is scattered in small collections about in the medullary tissue. Or else the pus is not evident; but the medullary tissue is spongy looking, red and tumid; it may then be very difficult to decide upon its state even by microscopic examination. For we know that the reddish marrow of cancellous tissue naturally contains more or less of a substance full of lymphoid cells not unlike pus-cells. These cells are often reddish, and not long ago Dr. Naumann advanced the hypothesis that blood-cells arise in the marrow, and that these red lymphoid cells are really blood-cells in process of development. Besides the ambiguity so arising, there is generally so much oil in the specimens that we are much impeded in our examination by fat-globules, and hence it requires a very careful inspection of all the characters of the case to come to a secure conclusion on the state of the bone. Sometimes there is lymph or pus in some parts, while in others the spongy reddening of the tissue prevails, the former parts being of a sulphur colour, the latter contrasting sharply by the brightness of their redness. We once met with a case of this acute diffused osteo-myelitis as the only secondary suppuration in a case of pyæmia from a carbuncle in the back, so that it may be the effect as well as the cause of pyæmia. When the inflammation is in this acute form the bone often dies throughout, and so may be found lying denuded of its periosteum.

But in some of the cases the inflammation is not diffused so freely, but limits itself to the parts of bone around the medullary canal. We see this frequently enough revealed in the form of a sequestrum from the interior of a bone after amputation. When the bone has been involved in acute inflammation of the stump and the suppuration has extended up the bone in the form of osteo-myelitis, it goes higher up the interior than the exterior of the bone, and the consequence is that the interior of the bone dies to a greater height than the exterior, and when the *exfoliation* of the dead portion is complete and the *sequestrum* separates, there is a long piece withdrawn from the interior of the bone in continuity with the piece which lay exposed at the dead end of the bone. This extension of suppurative inflammation up the medullary cavity is always dangerous, tending, as we said, to produce pyæmia, though the frequent separation of these conical sequestra shows that pyæmia is not a necessary result of such myelitis. Whether pyæmia shall occur or no in these cases is not altogether determined by the extent of the osteo-myelitis. Fatal pyæmia often is associated with a very little myelitis.

*Chronic diffused osteo-myelitis.*—Sometimes we find the whole of a bone enlarged, and its texture either greatly condensed and indurated, or with the cancellous tissue yet open and spongy, but full of lymph in various stages of disintegration or ossification, the indurated and lymph-charged parts prevailing more or less in different instances. We have seen specimens of the femur diseased in this way, where there was no part actually dead. The femur appears to be more liable than other bones to this affection. In some cases we meet with the same condition in a rather more acute state, the shaft of the bone being red and soft, and the soft cancellous bone being infiltrated with lymph more or less resembling pus. In all these cases the surface of the bone and periosteum shares in the disease. The course of this affection is, however, very different from the acute osteo-myelitis. The cases generally are of several years' duration. The soft parts about these bones are greatly altered, the periosteum united with the muscles, and these largely changed to fibrous tissue. Some examples may have been syphilitic, but it has not been possible to prove such a cause in others of them. A lower degree of ostitis is sometimes met with, especially in the cranium, and especially as the result of injuries. Thus, we may see the whole calvaria covered with a number of bony granulations, while it is thick, dense, and has lost its diploë. The soft parts, within and without, participate in the inflammatory irritation, which thus may deserve the name cephalitis. The same is true of the spine: signs of disease respectively similar are not uncommon after severe injuries to the spine, but the anatomical effects are not so well known; we have found the vertebræ thick and rough in such cases. The history of injury is often remote, so that the suspicion of a constitutional cause, such as syphilis or rheumatism, cannot be altogether set aside.

*Circumscribed osteo-myelitis.*—This also occurs both as an acute and as a chronic disease, though the distinction is less significant here, as the cases which arise acutely generally run a prolonged course, and so come ultimately to much the same character as those which commence gradually. Acute circumscribed osteo-myelitis is a rare affection, many well-described cases of which, however, are to be met with. The disease produces intense local pain, and after a period of great suffering, aggravated in nightly exacerbations, pus is found to have formed, being discovered either by the surgeon's exploration or by the spontaneous appearance of a swelling. Circumscribed chronic osteo-myelitis differs only in beginning more slowly and insidiously. Some of the cases recover after evacuation of pus from the bone by drilling or trephining; when the course is adverse, and the diseased parts come under anatomical observation, the case has generally lasted a long time, and the limb affected has been removed after many patient endeavours to obtain a cure. We then find the diseased part, which is generally the articular end of a long bone, thickened, swollen, reddened, and a little softened ; its cancelli full of lymph or pus, or else hardened and condensed by the ossification of the new product within its cancelli. This state rarely proves sufficient to render the limb hopeless, unless there be also present either a *chronic abscess* in the enlarged and inflamed bone, or else necrotic fragments lodged within the bone. This is thickened and diseased at the spot, with one or more sinuses, leading, perhaps, into a large joint, as well as to the surface of the limb. Perforating sinuses may be found without necrosis, as in an interesting case by Mr. Holmes, who believes that some sinuses may form by deep ulcers extending from the articular surface of the bone. The periosteum is often a great deal involved, so that it strips readily, or is already separated, and under it, or more frequently on the articular surface, a view of the bone may disclose eroded patches of ulceration, forming *simple ulcer* of bone, or *caries simplex* as it is sometimes called. This must be distinguished from true or fungous caries by the comparative firmness of the bone upon which the simple ulcers occur, a thin layer of condensed bone being beneath the ulcerous surface, and by the absence of the characteristic, more or less widely extended, caseous infiltration of true caries. We, however, find the limits that distinguish ulceration and caries of bone naturally vague, and in some cases it is very much a matter of choice which view we take of the nature of the disease. Indeed, as we shall presently see, caries is nothing else than a cachectic ulcerative osteo-myelitis, so that its relation to other obstinate forms of chronic ulcerative osteo-myelitis which we are now describing will naturally be very close in many cases.

When the small bones of the wrist and tarsus are affected with chronic inflammation, the disease has a course very similar to that of osteo-myelitis of the ends of the long bones. But there is often even a greater difficulty in distinguishing such simple chronic inflammation from caries, especially as the mechanical attrition of the ulcerous surfaces of the articular ends may wear away the bone, and waste it into a very deformed shape : yet there is no doubt that many cases of chronic tarsal and carpal disease are simply inflammatory, their obstinacy being due to the death of the inflamed portion of the bone. Necrosis of bone is frequently caused by the inflammation becoming acute, and then we often find, on examining the diseased parts, that the bone, where not necrosed, is in a healthy condition, and covered with strong granulations, or, perhaps, simply indurated by partial ossification of new products within it. In such cases the removal of the dead bone, when it is possible, forthwith alters the whole course of the malady, and repair at once commences steadily, progressing to entire cure, with or without reproduction of the bone, according as the periosteum has or has not escaped destruction. On the other hand, a small patch of caries has often been known to delay or wholly prevent the hoped-for cure after successful removal of dead tarsal bones or resection of the articular ends of long bones.

The purest examples of simple chronic osteo-myelitis are those that result from severe injuries, such as gunshot wounds or compound fractures. Many cases have occurred wherein, long after the healing of injuries of these kinds, inflammation has again appeared in the bone, leading to the death of a part of it. Sometimes minor degrees of injury, such as occur in children from blows on the head or falling from the erect posture, have produced ostitis of the cranium, with necrosis and separation of considerable portions of the calvaria. We have before described the more diffused effects of such injuries. Periostitis and caries are also sometimes met with after typhoid fever, and some other disease.

*Abscess* within a bone, at least in the form of a considerable collection of pus without necrosis, is rare; it occurs generally in the cancellous tissue of the long bones, especially of the femur and tibia, and may be contained in a distinct membrane. The bone is in typical cases expanded by an extension of the suppuration in the internal tissue, with a simultaneous formation of new bone under the periosteum, as the latter is pushed out by the increasing abscess. In this way the end of the bone may enlarge to a great size, and such was probably the mode of origin of some of those large cavities in the ends of dried bones which are to be found in Museums under the old and absurd name of *spina ventosa.*

*Ostitis.*—We have described periostitis and osteo-myelitis, so far, as separate affections, but in most cases the inflammation implicates both the periosteal surface and the medullary tissue at the same time. The result then is simply a sum of the effects of periostitis and osteo-myelitis. We have frequently alluded to this combination of superficial and deep inflammation, but it requires to be specially mentioned that the name *ostitis* is given to such an implication of the whole substance of the bone in inflammation.

Great increase of the medulla is said to occur in some cases of leuchæmia, and we have an example in the Museum supposed to show this. We have also a specimen where the medulla is of a reddish colour in a case of purpura.

*Syphilitic inflammation of bone.*—Syphilis produces periostitis, osteo-myelitis, ostitis, caries, and necrosis, in short every one of the kinds of inflammatory affection to which the bones are liable. In this the syphilitic bone affections resemble the syphilitic diseases of the skin, which we know are equally manifold in their forms, exhibiting all the simple kinds of cutaneous disease modified through certain peculiarities of colour, &c. On the cranium, for example, many are annular, excavatory, or have round edges. The effects of syphilis on the bones are often characteristic and recognisable. The leading feature is that which marks all tertiary syphilitic lesions, namely, the limited area of the diseased change, and its productive character. Circumscribed thickenings, and low elevations on the shafts or flat surfaces of bones, are the most common effects of syphilis. These are well known under the name of *nodes*, but in some cases the whole bone or many bones, even to the whole of the skeleton, may be affected. The disease also assumes all degrees of severity, and extensive necrosis may occur, though fortunately it is not nearly so common now as formerly, when the treatment of syphilis was fatally active. Even now necrosis of the bone may be induced by unnecessary incisions into fluctuating nodes, and a curable disease thus may be made almost or quite incurable. When necrosis occurs through syphilitic inflammation, the bone often presents peculiarities by which the nature of the disease may be recognised. This happens through the fact that the disease generally progresses for some time in the affected spot before necrosis takes place. In the first stage of syphilitic ostitis, as usual in all ostitis, the bone is rarefied by increase of the proliferating medulla at the expense of the *tela ossea*; thus the cancelli are enlarged, and the superficial hard layer is rendered porous. This process goes on until the affected part becomes more or less spongy before the necrosis comes to pass. Besides this, the dead piece of bone generally has a rounded figure with indented edges. This porous, round, indented sequestrum is very different from the smooth sequestrum of common inflammation. Such necrosis is generally seen on the cranium, and there also we usually meet with another characteristic effect of the same process of absorption of bone in the form of depressed patches. The slow, circumscribed, rarefying ostitis here, instead of leading to the death of the bone, removes the *tela ossea* and softens the bone, so that the superficial layers of it sink in over the space affected, forming a circumscribed depression, marked with radiating vascular grooves. The depressions from the two tables of the skull may meet, perforating the bone completely. Under these circumstances the matter which fills up the open cancelli is not pus, but granulation tissue, so that no suppuration accompanies it, hence the name *caries sicca*, by which it is known. Another peculiar form of syphilitic bone disease, also generally found on the calvaria, consists in the erosion of the surface along curiously curved sinuous outlines, surrounding and partly enclosing areas, under the surface of which enclosed parts the erosion extends so as to throw off a thin scale of the bone. Here, too, the substance that forms in the groove is granulation tissue. We have known it extend through the vascular openings in the skull, along the vessels, and reach the sinuses of the dura mater, causing remarkable thickening of these, and in one case entire occlusion of them. After the more active rarefying stage of syphilitic bone disease has passed off, there is always a rapid and effective hypertrophic tendency in and around the affected part, so that syphilitic bones are always heavy and hard. In caries sicca and sinuous erosion the induration is generally not accompanied by periosteal elevations.

*Hereditary syphilitic disease of infants.*—It is believed by many that all the bones of the skeleton may be affected in those who are the subjects of inherited syphilis, and that an inflammatory process in their surface and substance will cause their thickening and softening. In our Museum is a very old preparation of a skeleton of a child, showing enlargement and caries of the condyles of the humerus, which has been regarded as syphilitic; and Mr. Parker has placed in our Museum the thigh-bone of an infant, which he considers to be characteristically affected. There is a new growth of bone around the shaft, and so superimposed that there is a delicate layer of cancellous structure between them. Some years ago Mr. Sutton took to the Pathological Society the bones of a child which had undergone spontaneous fracture, and some of them separated at their epiphysis. The child had sores upon its body, and lost its nose by ulceration. All these cases would formerly have been called strumous.

M. Parrot, of Paris, having given much time to the subject, concluded that all the bones might be attacked. It was more especially the lower ends of the long bones which were liable to be affected, either by the production of a gelatiniform porous substance or a harder cretaceous ossifying material. As regards the *cranium* he clearly showed that a very characteristic change occurred in syphilitic children. This was the production of four bosses or prominences on the top of the head surrounding the anterior fontanelle,—two on the frontal and two on the parietal bones. These four prominences are well marked on passing the hand over the head. They are not due merely to a periostitis, but to a thickening of the bone or production of new osteophytic layers. This may be seen in the specimen in our Museum. The description was subsequently confirmed by the investigations of Dr. Barlow.

*Caries.*—We limit the term *true caries* to what is called *caries fungosa* by those authors who call simple ulcer of bone caries simplex. *Caries fungosa*, or true *caries*, is that disease of a bone which corresponds to scrofulous inflammation of the soft parts, and it shares all the obscurity of this class of diseases. By scrofula we mean the slow caseous inflammations that arise without sufficient extrinsic cause, and do not show that tendency to recovery which characterises inflammation from injuries in healthy subjects.

The word caries is in constant use, and yet there is, perhaps, no word in the whole of Pathology which is of less certain application. Thus, Sir J. Paget gives it no special place in his work, describing the changes others call caries under the name of tubercle of bone, while the nomenclature issued by the Royal College of Physicians gives caries as one disease, and scrofulous or tuberculous disease of the bone as another disease.

The best way to realise the question for settlement will be to ascertain what it is that is practically called caries by those who use that term. We find the state pretty well understood and agreed upon. The chief characters of the disease called true caries are these:—1. The bone is softer than natural, being rarefied by the transformation of its *tela ossea* to medulla, by decalcification and multiplication of the lacunar cells. 2. Its medullary tissue is charged with inflammatory matter disposed in patches in the expanded cancelli, the inflammatory matter having more or less of a caseous quality in the centres of the patches, and so resembling the usual products of scrofulous inflammation, which are called "crude tubercle." 3. The bony substance is not only soft, but itself undergoes fatty degeneration of its cells, and breaks down, yielding more or fewer small fragments, crumbling away spontaneously by what has been called "molecular necrosis." 4. The bone has a bare surface either on the periosteal or articular face, or else towards the cavity of an abscess, or of a chasm containing dead bone. 5. This surface is irregular and eroded in its appearance, and shows more or less of weak, pale, flabby granulations, which may be large and tender, as in caries of the internal ear, when they may form polypoid projections into the meatus exquisitely sore. 6. The discharge, instead of being purulent, is more or less watery and sanious, perhaps containing blood. Its effect upon the soft parts it passes over is a greater irritation than that caused by "laudable pus." 7. The surrounding bone, instead of being thickened, as it always is in simple ulcerative disease of the bones, is healthy, or in other cases it is greasy, being filled with an unnatural amount of fat, an interstitial absorption of the bony texture progressing to such an extent as often to make the bone yield under the pressure of a finger. When the bone shows these characters it is said to be carious, and there is, we think, no doubt about the matter as far as this. But the difficulty comes when the question arises whether this state constitutes a peculiar and specific disease of the bone, some thinking that any unhealthy ulceration, due to syphilis, scrofula, or even general weakness from any cause, is equally caries, while others say that caries of bone is quite a peculiar condition, and may be met with in persons who exhibit no constitutional weakness at all.

We shall find, however, that this kind of dispute is not at all peculiar to the question as to the nature of caries. It equally applies in exactly the same terms to all those other instances of chronic suppuration which arise from insufficient extrinsic cause, and tend little to heal, and which are called by the general and very useful name "scrofulous." In so-called "scrofulous" suppuration of the kidney it is equally true that we are not always able to show the described constitutional signs of scrofula. We think it is better to abandon these described signs of scrofula altogether, for their range and application are hopelessly inexact, and to limit the meaning of the term scrofula to a state of tissue that reveals abnormal vulnerability and abnormal tenacity of injury, showing these characters by chronic suppurative destruction of the affected part without sufficient of the reparative formative process by which the suppurative inflammations of healthy persons are terminated and healed. The intense obstinacy of caries has favoured the disposition to ascribe to it a specific and peculiar nature. The impossibility of cure has been thought to be a specific distinction of true caries from common inflammation. When, however, we consider the conditions of bone in regard of the process by which ulcers heal, we can understand how imperfections in the formative activity in ulcers should in bone be of far greater consequence in impeding cure than in the case of the soft tissues. We know that in the healing of ordinary ulcers the parts are restored by two processes, the first of which is the production of new matter to fill the space, the second is the closing up of the parts around, either by their swelling or by the contraction of the new tissue formed in the other process. Now, of these two processes the second is often the more efficient, and is quite necessary if any considerable loss of tissue has occurred. We are, indeed, too familiar with the unfortunate results that arise when only the first process is available, and the second is prevented by the state of the parietes of the ulcerous space. Thus, it is to the rigid and unyielding walls of the cavities they occur in that the obstinacy of phthisical vomicæ and of anal fistulæ is due, so that when a surgical operation enables the walls of an anal fissure to close in upon each other the healing of the sinus is soon achieved. If, now, we apply these reflections to the case of carious bone we see that the bony tissue is too rigid to allow of closure of the ulcer by collapsing around the loss of substance. No closure by swelling or collapsing of parts around can occur, and hence all the obstinacy of fistulous disease is to be expected, especially as the formative process is enfeebled and impeded by the resistance which the hard *tela ossea* offers to the free growth and nourishment of the granulations themselves. We have already said that in many cases of obstinate disease of the bones, leading ultimately to resection or amputation, the state of true caries is not present, the chronic inflammation of the bone not being accompanied by caseous softening, as in caries, but by thickening and induration. The obstinacy will then be found due to the existence of a sinus or abscess in the bone or a piece of necrosed bone embedded in it, the inability to heal being due to the mechanical impediments to the closure of the cavity. As to the so-called syphilitic caries we have always found that it differs essentially from the true caries of chronic scrofulous inflammation in this, that the bone about the disease is thickened and not rarefied, as in the chronic scrofulous disease. Indeed, in syphilitic caries of the skull a very great increase of weight of the whole skull is commonly present, whereas the whole of the dried femur in cases of true carious disease may not weigh more than two fifths or two thirds the normal weight of the bone. Caries usually affects the short bones of the wrist and tarsus, the vertebral column, or the ends of the long bones. The effects of it in these different parts are rendered different through the various anatomical relations and physiological circumstances of the bone affected. Thus, the small bones of the wrist or tarsus will undergo caries for a greater or less time, and so be reduced to four fifths, three fourths, or two thirds of their proper size, and then very often comes a new result, for the disease, extending all around the bone through its smallness, kills it by separating it from its periosteum and so from its vascular supply. For in carious inflammation, unlike healthy chronic inflammation, no new vessels are formed to connect parts separated by the lymph that has collected between them. Thus, caries of the small bones is after a while usually complicated with necrosis. The facts often come before the surgeon apparently in the reverse order in this way. He operates for the removal of a piece of dead bone from the wrist or ankle of a small child; afterwards the case does badly and amputation is required, when it is found that the necrosis was only a part of the disease, the natural result of a caries which had spread so as to involve the entire surface of the bone, or so much of it that the whole bone had perished, the caries afterwards spreading amongst the other bones, and going on, perhaps, towards destroying others in the same way as that which had been removed. Caries of long bones, on the other hand, can implicate only a relatively small extent of surface, and hence is oftener uncomplicated by necrosis, since, as the disease extends slowly around the bone, the supply from within the medullary canal has time to enlarge itself and meet the new demand. Caries very rarely affects the shafts of the long bones; it is usually limited to the epiphyses. When caries attacks the bones of the extremities the products of inflammation may be long retained or even reabsorbed, being in small quantity, and consisting only of granulation tissue without pus. But when pus is freely formed the abscess is readily opened, and the pus is then found to be watery with flocculi of caseous matter suspended in it. Analysis shows such pus to contain excess of lime salts, and often small fragments of bone may be detected in it. But it is to be noted that abscesses and sinuses around carious bones do not always reach the bone or joint. Some abscesses arise by local suppuration in the inflamed tissues outside the joints, forming circumscribed abscesses, which, being opened, form sinuses.

Some surgeons have been content to call caries an inflammation of the bone with the formation of granulations, made up of a mass of cells, and an absorption of the old bone. Any swollen granulating bone permeated with pus they would thus call caries.

There has been a tendency of late, however, to revive the opinion that *tubercle* is the primary starting-point in many chronic diseases of the bones and joints which were formerly called scrofulous. Besides the authority of some German pathologists we have the opinion expressed in its favour by some of our modern hospital surgeons as Croft, Macnamara, and Barker. They describe how a deposit of tubercle may occur in the bones, ending in caries, or in the head of the bone, giving rise to tubercular disease of the joint. Small deposits may, it is said, be seen, for example, in the neck or head of the femur composed of small cells, giant-cells, and sometimes bacilli. In these a yellow caseation takes place leading to softening, suppuration, and the usual results.

The so-called white swelling of joints is thus tubercular in its origin, beginning sometimes in the bone, and sometimes in the synovial membrane,—in the majority of cases in the former. Tubercle is also described as occurring in the vertebræ and the bones of the tarsus, and less commonly in a diffused form in the shafts of the bones.

*Caries of the spine* offers a new condition from the pressure of the weight above the carious part, which necessarily tends to bend the spinal column, and as the bodies are the parts of the vertebræ affected, while the spinous processes, &c., generally escape, the bend takes a forward direction, an angle with backward projection being produced by the sinking of the bodies of the vertebræ above to fill the hollow caused by the wasting and removal of the bodies at the diseased spot. This angular curvature is called *Kyphosis.*

The disease in the spine is also peculiar through the presence of the intervertebral substance. That disease of the spine often shows itself first in the intervertebral substance as insisted on by Mr. William Adams is fully borne out by our experience. It may not yet be certain whether these cases should be regarded as a special kind of disease, distinct from the true scrofulous caries, which, according to some authorities, always begins in the bones. Certainly we have frequently met with cases where the intervertebral substances were specially affected, yet the bone was decidedly carious. And then it was clear that the disease in the bone had been propagated from the intervertebral substance; for instance, we may often find a vertebra apparently destroyed in its middle by disease, but on closer examination the two portions which appear to be the remains of one are found to belong to two separate bones, the disease having commenced between them. This has happened so frequently that we cannot agree in Rindfleisch's statement that in caries of the spine the intervertebral substance is either not at all implicated or only late in the course of the disease. But it is probable that in many cases of so-called caries of the spine beginning thus the disease is not scrofulous. Indeed, the great mobility of the spine, and the pressure to which its parts are subjected, explain the unhealing quality of many neglected inflammations due originally to a strain or blow, without our being obliged in explanation to have recourse to any peculiarity of constitution on the part of the patient. But we think the character of the morbid process is very different in the two classes of cases, of which one is typified by the spinal disease of weakly scrofulous children, and the other by the spinal disease of otherwise healthy adults. In the scrofulous cases the bones are soft and little or no sign of repair is present, the conditions being just such as we have described as characteristic of caries. On the other hand, in cases which occur from injury in healthier subjects the bones are indurated, and new bone is formed around the seat of disease, and the tendency to repair may be so effective as to end in complete ankylosis of the bones in their new angular position, causing the hunch-backed figures we not unfrequently meet with. Such recovery is, indeed, favoured by the angular curvature bringing the bones together within reach of each other. All stages of the destructive and reparative processes in bones may be seen on our shelves. In one example, which was taken from the body of a strong and previously healthy man, there was not the slightest indication of scrofula, and, according to the history, no doubt the disease began in the intervertebral substance. The man strained his back in carrying a weight, the intervertebral substance was slightly lacerated, some lymph was thrown out, and subsequently a gradual destruction of the whole cartilage took place, and at last the adjacent surfaces of the bones were involved. We may see, then, in some of our specimens a yellow soft matter, affecting only the intervertebral substance; in others, a destruction of this substance with the adjacent bones; and in others, numerous vertebræ altogether destroyed. We see also in these specimens how repair takes place. In some instances the fragments of bone are held together by ligamentous tissue, and in others they are firmly conglomerated into one irregular mass of bone. An extreme angular curvature necessarily results from the vertebræ thus falling together. We may observe how remarkably the medulla follows the curvature of the bone, and how it escapes mischief in numerous cases. *Lumbar* and *Psoas abscess* are shown very imperfectly in our dried preparations. They usually result from caries of spine. In one case, where there is disease between the fifth and sixth dorsal vertebræ, a lumbar abscess existed on one side and a psoas on the other. Sometimes the articular processes are affected, or the adjoining ribs are involved in the caries.

The question whether the disease begins in the bone or in the cartilage has taken a fresh interest since the revival of the opinion of the frequent occurrence of tubercle in bone; many now making the starting-point in the bone. A difficulty in tracing the origin of caries arises from the fact that the inflammatory process begins between the cartilage and bone, invading both and proceeding towards one or the other in different cases. In some of our examples we see the cartilages almost gone, and the bodies nearly meeting.

Disease of the *Odontoid process* may occur as a separate affection. In one of our specimens will be seen a remarkable instance of this process becoming carious and making its way out through the pharynx, the patient completely recovering.

Disease between the first two vertebræ, the atlas and the occiput, is not uncommon, and results in ankylosis, numerous specimens of which can be seen on our shelves. In all our specimens of disease of the *Cervical vertebræ* we see that the abscesses which accompanied them opened into the pharynx.

Caries of the cranium we have already alluded to, but there are other specimens which show rather a simple erosion as effects of lupus or facial cancer. We perceive there is no new deposit of bone around the decaying edges, as in caries, but a simple erosion of the structure.

*Necrosis.*—We have frequently had occasion to mention necrosis or the death of bone as a consequence of severe inflammation of its medulla or periosteum. This is the most common cause of necrosis, but it is met with also as a result of accidents. Fragments may be entirely separated from their connections, and hence may perish; more frequently the periosteum is stripped off the bone, and perhaps injured or destroyed at the same time, or the bone is exposed to the air through the wound in compound fracture. Under either of these circumstances there may be any degree of inflammatory complication co-operating with the injury to destroy more or less of the bone. Thus, a simple inflammatory action may be set up and a speedy cure result; or the bone may become ulcerated in the same way as the soft parts, and after a more lengthened period form granulations and heal; or, again, if a large portion of the periosteum be destroyed, the bone is likely to die, for, the periosteum being removed, the vascular supply is cut off, and the exposed surface mortifies, the bone becoming white and subsequently black. To a certain depth only does this change occur, for the deeper seated portion of shaft being well supplied by the medullary membrane and adjacent vessels, a line of separation ensues between the part thus nourished and the exterior.

Inflammatory granulation tissue thus forms along this line by decalcification of the bone in it and production of the granulations from the soft substance thus set free, so that in the course of some weeks the dead portion becomes detached, leaving the living raw surface below covered with vascular granulations; these then become changed partly into new bone and partly into a new periosteum, which forms, with some fibrous tissue and the adjacent soft parts, a cicatrix. A small scale of bone may thus come off, or, if some inches of the shaft be exposed, a portion extending to the medulla may come away; if more, of course there is danger of death to the whole bone. This process of separation of a portion of dead bone is called *exfoliation;* it may be well seen in these specimens of diseased crania, where, owing to the distinct vascular supply of the interior and exterior of the skull, large portions of the external table have come away without interfering with the deeper portions; we here see a large piece of bone, the size of the palm of the hand, in the process of separation, and on looking at it we also see how it is bevelled off, so that the external surface is much larger than the internal. Even in this specimen, where necrosis has resulted from severe injury, involving the whole thickness of the skull, the internal table is only brought away from a single spot in the centre of this large piece, which has nearly all come from the external table.

We so far have spoken of the dead bone exfoliating and coming away as if this were a matter easily accomplished. But, unfortunately, this is far from being the case; indeed, it is the great difficulty that attends removal of the dead bone which has drawn so much attention and brought so many long names to the description of necrosis and of the parts concerned in it. When the piece of bone is separated or *exfoliated* it is called a *sequestrum.* This sequestrum generally does not lie free among soft parts, it is more or less enclosed in bone. This enclosing bone may be either the surrounding parts of the original bone, as when the portion which died was deep seated within the natural bone, or else, if the necrosis was superficial, it will be new bone, which arises in the following way. The periosteum, whose separation is the cause of the death of the bone, is thrust in the separation to some distance from the bone, being separated from it by blood or pus, according as the disease was due to injury or inflammation. The deeper layer of periosteum which immediately encloses the bone is softer than the rest of the periosteum; it has well been called the *cambium layer*, since, like the cambium in a tree, it is the seat of the active growth; it also has a soft consistence, so that it tears readily. This cambium layer has a very great ossifying power, and when displaced from around the bone it begins to form bone in its new situation as soon as destructive inflammation subsides. Thus, it creates a case of bone around the dead part of the shaft. This case of new bone is called the *capsule.* The new formation of bone always extends some distance beyond the necrosed part of the shaft; it goes on under the periosteum of the remaining part of the old bone at both ends, uniting there with the old bone. Thus, the new periosteal capsule of bone is made continuous with the remaining parts of the original bone. It is not quite a complete sheath, but shows a few round openings, which lead through it inwards to the space between it and the sequestrum, and outwards into those fistulous channels which conduct the discharge to the surface. When such a bone is dried these openings, called *cloacæ*, are found as round orifices in the capsule of bone, with everted, rather prominent edges. Surgeons are in the habit of passing probes through these cloacæ and feeling for loose bone within, which, if found, is removed if that be practicable. We may here mention that, in a specimen of a tibia, where the new bone is bent, it is probable that the attempt to remove the old bone was made too early, for it is necessary that it should remain a sufficient length of time as a model for the new bone to be formed upon; if not, the latter, being soft, is acted on by the muscles and becomes distorted; the same happens in the lower jaw if necrotic bone be removed at too early a period. It will be explained in the surgical lectures at what period interference is necessary; but we show these specimens that it may be understood how the new bone is modelled upon the old. In young persons necrosis of long bones affects the shaft alone, and, the epiphyses remaining, the joint escapes; but this is not universally the case, and in adults the disease is very likely to continue to the extreme ends of the bones; in such instances, with the joint involved, there can be little hope of repair, but we think it possible for an epiphysis to be renewed, though with ankylosis of the joint, as seen in the specimen.

There are some special circumstances under which necrosis occurs which require to be known. Those engaged in the manufacture of lucifer-matches suffer necrosis of the jaw-bones. The mischief is set up through the action of the fumes of phosphorus; it is called in the match trade "the match-maker's disease." It commences along the alveolar border, perhaps in the seat of decayed teeth. The disease spreads slowly, and is usually accompanied by a free formation of spongy new bone on the margin of the necrotic portion.

In the Clinical Report to be found in the 'Guy's Hospital Reports' for 1847 we allude to a case which seems to be the first recorded instance of this affection. After speaking of diseases of the jaws, we say, "One case occurred in a lucifer-match maker with suppuration and exfoliation of the bone. The disease had previously been noticed to be not uncommon in those working in phosphorus." Mr. Hutchinson reported the case of a lady who had necrosis of the lower jaw from taking phosphorus pills.

Another remarkable special instance of necrosis is in the case of some children after exanthems: a portion of the alveolus of the jaw-bone perishes and separates, bearing with it the milk tooth, and sac of the permanent tooth; no more of the jaw comes away than is required to liberate the teeth.

**Injury.**—The bones pay a tribute for their firmness in their liability to fracture. In children and young persons the fracture may pass through the epiphysial cartilage, so that separation of epiphysis results. The bones may be perforated or crushed by gunshot, or cut with sabres, &c. Sometimes a mere bruising of bone, especially of the cranium, when little is feared from it, may set up suppurative inflammation, leading to acute necrosis, pyæmia, and death.

*Fracture.*—When the broken bone is exposed in a wound leading to the surface the fracture is called *compound*, otherwise when there is no wound exposing the bone the fracture is called *simple*. This distinction corresponds to a very important difference, since the healing of simple fracture is a very simple process, unattended with suppuration or serious inflammation, while the healing of compound fracture is a complex process, much slower, often attended with suppuration and granulation, and then involving no little danger of osteo-myelitis, with its terrible follower, pyæmia.

In the case of simple fracture a soft matter forms around the ends of the bones and ossifies, thus uniting them together. When the ends override each other, or are not kept steady, a large quantity of this ossifying material is rapidly produced, so as to form a large mass around the fracture; but if the apposition of the ends be even and close, the amount formed is small, so that the swelling may not be detectable. Whence comes this ossifying matter? Whenever a bone is broken there is some bleeding from the bone itself, but more especially from vessels torn in the sharp jerk that accompanies the breaking of the bone. It was formerly a much contested question whether the blood thus poured out undergoes ossification, and the teaching of late has generally decided in the negative. Greig Smith, however, maintains that the effused blood forms an important element in the process of repair; that it plugs up the fissures, and then becomes organised.

It is generally thought that the new ossifying substance is formed from the periosteum principally, but partly also from the medulla of the bone. If the bones be not well placed together, other tissues, where they are continuous with the periosteum, will take to forming the bony union, as though they were infected by its kind of activity. Whether the periosteum or the medulla produces the greater proportion in any case depends on the circumstances of the case. If the ends be left more or less overriding, so as to meet by their sides, then the periosteum becomes quickly much swollen and softened for two or three inches on each fragment. How much of this swelling occurs depends on how bad the position of the ends, and how unsteady their maintenance. The thickening is greatest at the end, and gradually diminishes up the shaft. The swollen periosteum of the fragments unites where they meet, and then a soft mass is found joining the broken ends. This mass is continuous, especially with the deep layer of periosteum, and it has essentially the same microscopic structure, but is more translucent, being in some parts very much like cartilage from the hyaline appearance of the intercellular substance, and the encapsulated look of the cells. Indeed, it sometimes is so like cartilage that the name cannot be denied to it, but at other times it is more fibrous. According to some a temporary cartilage must always precede the formation of new bone, but different views are now reconciled by calling the cells osteoblasts. This mass is called the "callus," or the *external callus*, as distinguished from the similar mass produced from the medullary tissue which closes the hollow broken end of the shaft, and is called the *internal callus*. It is also called *provisional callus*, because none of it ultimately remains unless it is wanted to secure continuity in the line of the shaft. The production of this swollen mass is exactly that process which we have described as "ossifying periostitis." Bone soon begins to appear in it in the form of spicules so arranged as to compose a sponge-like porous mass, in the channels of which are new vessels. This soft bony sponge commences about the third week, and during the next three or four months undergoes a modelling process, so that so much of it as is required to complete the shaft passes on to the formation of hard bone: this is done by the channels of its spongy tissue being filled up with concentric laminæ of bone, which narrow them to the proportions of Haversian canals, thus making hard bone; but the greater part of it is not needed for the continuity of the shaft, and it wastes away and is absorbed; so, too, any projecting ends of the fragments lose their prominence, and are rounded off until at last there is left no more than will complete the fragments into a column.

Meantime the fractured faces, on which of course the medulla is exposed, are undergoing corresponding changes more slowly and less largely. If the position be very good, little external callus appears; it may even not be detectable in a fractured tibia by examination with the finger; but the broken ends lying adjacent take on that form of action called *rarefying ostitis*, the lime salts disappear, and the cellular medullary tissue set free forms a spongy mass of callus that closes the end of the shaft, and meets with the corresponding mass in the other fragment. These masses unite with each other, so that the continuity can be at once restored by the ossification of this *internal callus*. Afterwards, when this callus has become thoroughly developed into ivory bone, a modelling process of atrophy reabsorbs that portion which filled up the marrow canal, so that in the end there may be scarcely a trace of it, and no sufficient evidence that a fracture ever existed. Thus the callus serves to maintain the ends in temporary union, performing the service of splints, and usually signifying the want or insufficiency of these. For provisional callus, as we have seen, is not necessary at all, and occurs mostly in those cases where perfect rest cannot be commanded. It is well seen in fractures of the bones of lower animals, made in experiments, from which, indeed, the description of the union of fracture has been too trustfully derived, leading to a too great prominence of the provisional callus in the history. For a visit to the accident ward will inform us that a fracture may at once unite without any provisional callus; we shall not fail to find some cases of fracture of the tibia where the line of union can be scarcely felt. On the other hand, if the part be not kept pretty steady during the few months required for the modelling of the bone into its proper form, the provisional callus will not disappear, but will continue in the form of a permanent node of hard bone. This is well seen in our skeleton of the hippopotamus. That must have been a very unfortunate, or, perhaps, a very reckless animal, for he has broken all his ribs, without favour to any, except that some are broken in several places. At every injured spot there is a large hard node; no doubt the want of rest, and all the necessary movement of the ribs in breathing, caused the excess of bone formation. It is not very uncommon to find such an excess even uniting two or three ribs together, if the injury has been severe. In this preparation of a fractured clavicle we see the large, spongy, external, temporary callus uniting the bones together. This spongy bone would have been absorbed, when the ends of the bone were firmly united, but not so the hard nodes of the hippopotamus' ribs; they would be permanent.

Mr. Jacobson, in describing the repair of fracture, says that at first the ends of the bones are embedded in the extravasated blood, and in four days this is partly absorbed and partly lost in the mass of neoplastic material supplied by the bone, periosteum, and other parts around. On the tenth day cartilaginous and bony formation are fast proceeding both as an external and internal callus, the newly-formed bone being soft, spongy, porous, and vascular. The callus is then absorbed, and the whole union is complete in four or five months. The process is much abridged in simple fractures with ends well adjusted; there is then seen only a narrow line of newly-ossified material between the fragments.

When the fracture is *compound*, suppuration sometimes occurs about the broken ends, and the healing of the bone is effected by granulations forming and ossifying, as in ulceration of the bones.

*False joint.*—When the fractured ends of a bone do not unite, a false joint is formed. We must remember, however, that it is quite the exception to have a new perfect joint produced; all we find is that the bones, which either are too far separated, or kept apart by other tissues, or are deficient in repairing power, form, instead of bone, only a ligamentous tissue, which allows free play between them. Thus, in a specimen of a humerus, where a fracture had failed to unite eight years before, the ends have become pointed, and firmly joined by a strong ligamentous band, but there are no structures such as would constitute a true joint. Probably where there is a broader surface of contact more of a joint is produced. Thus, in a specimen of forearm, the ends of the bones are surrounded by something like a capsular ligament, and within is a synovial sac and fluid.

Some seats or kinds of fracture have special points in their history to which we must draw attention. Thus, *fracture of the neck of the thigh-bone* is far from infrequent, occurring usually in an elderly person who has suffered a sharp fall, and is then found to be powerless in one leg. We recently had before us an example of this fracture on both sides in a man seventy-two years old, who had been knocked down by a cab. The case was deceptively like paraplegia. Much interest attends this form of fracture because it is very rarely, if ever, repaired by bone, and many reasons have been given for this. One is that all fractures in joints are seldom thus repaired, because the breach is filled merely by a synovial fluid, so that there is an absence of the soft textures which would assist in supplying the necessary pabulum. But the chief reason is the difficulty of keeping the parts of a joint in perfect apposition: this reason obtains in other joints, for in the olecranon osseous union is rare, and as regards the patella its bearing is particularly evident, for in vertical fracture of that bone, where there is no difficulty in preserving contact, an osseous union usually occurs, while its transverse fractures seldom unite by bone. This, however, can be obtained by dividing the ligaments which are attached to the patella, and uniting the portions together. Another reason for non-union in the particular case of the cervix femoris is that the nutrient artery of the neck is divided, so that the only supply of blood to the head of the bone is by the round ligament. Old age, too, the time in which this accident occurs, has been considered instrumental in the prevention of union; but of this there is great doubt, unless it be owing to the degenerative changes which might be previously going on in the neck of the bone, for union in other parts of the skeleton may take place in old people; and, on the other hand, a want of osseous union in the joint is observed in the young. For example, in a bone from a child who received a severe injury, and broke the upper part of the os femoris both within the capsule and without, the external fracture appears to have been repaired, but the inner shows no sign of cure. We see, by observing specimens of fractured cervix femoris, how little repair has advanced after several weeks or months, and if the parts have in any way united, it is only by some ligamentous tissue. Such a union is all that we can generally expect. In a specimen, which we obtained some time ago from a woman who fractured the neck of the bone five years before, there is good ligamentous union, and, besides this, some nodules of cartilage between the broken extremities. In all these cases we see how the faces of the fracture have suffered absorption, and the neck is thus considerably reduced in length. Amongst these specimens there are some which are labelled as doubtful instances of bony union; and the difficulty of giving a correct judgment is often very great, for this reason, that a change very similar to that which an injury might produce is caused by senile disease. An old person, for example, has been subject for a year or two to the changes, which, as we have shown, are so often present in the neck of the thigh-bone, and which produce a shortening of this part. If now, in consequence of such impairment of the joint, the person should fall, and owing to the bruising take to his bed, and a medical opinion be sought, the leg being found powerless and shortened, it might be thought that a fracture had occurred, and then, if the case be watched, and after a year or two a post-mortem examination take place, it is highly probable that the distorted appearance of the neck of the bone may somewhat resemble a united fracture; for if a section be made, the irregular form of the cancellous structure may display some fibres of bone corresponding in place to a supposed line of fracture; and if, moreover, the joint has been affected by chronic rheumatic arthritis, some new bone may have been formed in the surrounding parts. That form of fracture external to the capsule, in which the neck is driven into the broken trochanter, unites easily by new bone, which binds all the parts together. This injury is known as *impacted fracture.*

*Fracture of skull.*—This subject will be given in full in the surgical lectures, and we will therefore merely request a careful inspection of the specimens. We shall see that they can be divided into two classes of cases, which correspond with a similar practical division we meet with in the wards according to the nature of the injury and the chances of cure. The distinction is simply this, some are circumscribed, and some diffused, and the difference arises in this way, that in the one class of cases the injury is due to a sharp blow struck with a light weapon, and in the other to a heavy blow from a larger body moving more slowly. In the first kind of fractures the violence does not extend its area much beyond the spot immediately struck. In the other class of cases the injury is diffused over the whole skull and its contents. The difference between the two kinds of cases may be well illustrated by what we have heard of in discussions between the two schools of naval artillery. There are two systems, in one of which the object is to throw a small shot with high velocity, and so pierce the side of the vessel—this is known as the punching system; while, in the other, a large shot is thrown with low velocity, so as to strike heavily, and shake and rattle the timbers of the ship—this has been called the racking system. In just the same way, when a sharp blow is struck on the skull with a hammer or sabre, or with a bullet, the parts immediately touched alone receive the damage. In these cases, the injury being circumscribed, there is a fair chance of recovery by judicious treatment. It is to this class of cases that most of our numerous specimens of repaired fracture belong. But when a man falls from a height upon his head, or is struck by a falling beam, or a cart wheel, in short, when the momentum of the blow is due rather to mass than to velocity, the fracture then extends far beyond the point of contact of the injurious agent. It is interesting to trace out the conditions that then arise; what happens is generally this, that a fissure starts from the point struck, and runs towards the base of the skull pretty straightly, but with more or less comminution, according to the severity of the blow. The fissure usually runs through the temporal bone, making its way in front or behind the rocky part of the petrous bone, so as to pass into the middle or posterior fossa of the basis cranii. The reason of this is that the petrous bones, from their position, being hard thick masses nearly over the condyles, convey the force of impact from the spine, and throw too great a share of this force upon the parts with which they are continuous, so as to strain and break those parts. It is easy to see that, when the skull is compressed to bursting between the spine and the ground, there will be a leading tendency for the rent to occur in a line from the spine to the ground, *i. e.* from the base of the skull towards the vertex. If the fall is upon the front or back of the vertex, this leading tendency to burst along the direct line between the two points of pressure will induce the fissure to run down the frontal or occipital bone into the anterior or posterior fossa; on the other hand, if the fall be immediately on the vertex, the momentum of the body, acting through the spine, will drive in a rim of bone around the foramen magnum, as we here see.

When the fracture leads into the middle ear, and ruptures the tympanic membrane, or runs into the pharynx, then there is bleeding into the ear or throat, as well as frequently an escape of the cerebro-spinal fluid, or even of the brain tissues. The fracture is apt, in passing in front of the petrous bone, to tear through the line of the Eustachian tube, which is a weak line; or, in passing behind the petrous, it is apt to crack through the very slight partition between the jugular fossa and the tympanum; or, not unfrequently, the petro-occipital suture is partly forced, and the spongy end of the petrous broken across into the Eustachian tube. In either of these ways a road through the bone to the torn tympanic membrane is opened up; now, such violence is always accompanied by laceration of the dura mater and arachnoid. If the naked eye does not discern the rent, a lens will show the surface of the dura mater to be cracked in severe injuries of this kind. The visceral arachnoid is nearly sure to be also broken, and thus blood and cerebro-spinal fluid come out into, and then out from, the arachnoid space through the external ear. Or the fissure, passing through the roof of the pharynx, tears also perhaps the lamina cinerea, forming the floor of the third ventricle, so opening a route for the fluid down from the ventricles, through the bones, into the pharynx. In some remarkable cases, collected by Mr. Prescott Hewett, a fracture of the calvaria communicated with the lateral ventricle, and so the ventricular fluid came away directly through the wound. In a very interesting and remarkable case, related by Mr. Lucas in our 'Reports,' a child who fell from a height on to its head had a large swelling rapidly appear beneath the scalp. This consisted of cerebro-spinal fluid. The child recovered for a time, but afterwards died of meningitis, when a fissure was found in the skull communicating with the expanded descending cornu of the lateral ventricle. Again, we have shown it to be nearly certain that in the common fracture about the jugular foramen the pneumogastric nerve is dragged on enough to tear the very delicate wall of the lateral horn of the fourth ventricle, with which it is closely united; so that in these fractures the fluid that comes through the ear is directly derived from the fourth ventricle. It was Mr. Hilton who first in this country proved that the fluid that escapes from the ear in these cases is of the same nature as the cerebro-spinal fluid. There have been cases, however, where no fracture has been discovered, and it has then been thought that the watery discharge came from the ear itself.

Another point of interest in fractures of the skull is the so-called *contre-coup*, a name signifying the tendency of injuries to show themselves at a point opposite the part actually struck. So far as we have observed, we never see the skull broken at the opposite point; the fracture of the bone is always where the blow was received, and extends from that spot as already described. So, too, the bleeding outside the *dura mater* is always at the point struck; in fact, this bleeding is from the meningeal vessels torn in the separation of the bone. This is, practically, a very important point; the blood is found in the form of a hard clot between the dura mater and the bone. But, on the other hand, the injury to the brain differs in its seat according as the fracture is of the kind we have called circumscribed or diffused. If the fracture is circumscribed, the brain is injured at the seat of the fracture; and if it be exposed, a hernia cerebri will occur, generally with an abscess behind it; but if the fracture is of the diffused kind, then the injury to the brain is always at the parts opposite to the point struck; for instance, if the fall is on the left side of the back of the head, the brain is torn in front on the right side. The part of the brain then injured is generally the prominence of the middle lobe and also of the anterior lobe. The middle meningeal artery may be ruptured from a blow without fracture of the bone, but this is very rare.

It is necessary to notice how unequal are the circumstances of different parts of the cranium with respect to the liability to fracture. Thus the condyle of the lower jaw may be thrust up, and break the glenoid cavity, or even be thrust through it; or the nasal bones, in a fall on the nose, may conduct the shock, and thrust the parts of the ethmoid with which they articulate up into the cranial cavity; or the excessively thin roof of the orbits may be burst at a great distance from the blow: we have seen a piece of the size and shape of a shilling broken out of the roof of each orbit by a fall on the occiput. This was the only approach to *contre-coup* of the bones we have met with; or these thin orbital pates may be thrust through with a tobacco pipe or umbrella, when little notice has been taken of the wound through the eyelid. On the other hand, the frontal sinuses may be broken only in their superficial wall, and in the same way, through the presence of the diploë in other parts, the outer table only may be broken, the inner escaping. But more often a blow on the outer table breaks the inner table to a greater extent than the outer; this is because the vibrations are dispersed, radiating away from the spot struck, so as to widen the area of the force as it goes from the outer to the inner table. In rare instances the outer table is not broken, or scarce broken, and the inner table is considerably fractured. In children the skull may be bulged in to a considerable extent in consequence of a fall, producing a curious deformity, and the dura mater being adherent to the soft bones, such an injury may tear open the membranes and brain. Sometimes the fracture reaches a suture and forces it apart, so producing what is termed *diastasis* of the suture. A separation of the suture is nearly always attended by fracture. If this occurs in the sagittal suture, the longitudinal sinus may be torn. In a specimen which we recently met with a Wormian bone in the angle of the lambdoid suture had been dislocated, and turned half round, so that its pointed end stuck into the longitudinal sinus, causing fatal hæmorrhage; and fragments may lacerate the carotid, causing death by hæmorrhage, or by suffocation by the blood passing into the lung. Indeed, if we were to mention all the curiosities of fracture of the skull, we should not be able to bring this lecture to an end. When circumscribed fractures are severe and even comminuted, portions of the bone may be removed and yet recovery take place; and even in bad cases of diffused fracture, when the fissure reaches the base of the skull, recovery may result, though this is much more rare, as we have already said.

We possess several portions of skull, picked up from the field of battle, showing very severe injuries, which have been repaired, and effectually repaired, as proved by the subjects of them having been able to engage in service again. These long fissures have, no doubt, been caused by sabre wounds, and, though the inner table is reached, recovery has taken place. In one, where the bone has been comminuted, but portions not removed, a union has occurred between the pieces. Where there is actual loss of bone, as from trephining, repair does not occur; but the dura mater on one side, and the scalp with the new cicatrix on the other, become united into a firm membrane for the protection of the brain. In another specimen, where there has been loss of bone, but whether from injury or disease is not very clear, the membrane taking its place is seen to have a few spicules of osseous matter shooting from the inner table, and a few isolated deposits in its centre.

*Fracture of the spine.*—In fracture of the spine the line of fracture takes a *direction* downwards and forwards, and generally implicates at least two vertebræ with the intervertebral substance between them. Sometimes the rent traverses the intervertebral substance only, or, more frequently, separates this from the bone, in which case the injury is called *dislocation of the spine*, and is distinguished from fracture. But the difference is of no importance, and, so far as we have seen, the more carefully the injured spine is examined the more surely is some injury of the bone discovered. It occurs more commonly from violent bending of the neck rather than by direct injury. In the *cervical* region, however, generally at the fifth vertebra, the intervertebral substance may be torn through without breach of the bone, and perhaps the same may happen in the *lumbar* region also. In the *dorsal* region the interlocking of the processes of the vertebræ is so close that there cannot be a dislocation without fracture. It will easily be seen that no considerable displacement of the upper on the lower fragment could occur without serious danger to the contents of the spinal canal; and, indeed, in most fractures of the spine there is a considerable displacement in the moment of the injury, although the bones are restored almost to their natural position afterwards by the action of the muscles. Hence the *spinal cord* suffers more or less severe injury, even to complete severance. But if the fracture is below the first lumbar vertebra, so that the nerves forming the *cauda equina* are implicated, these more commonly escape by reason of their comparative toughness and mobility. Hence fractures of the lumbar vertebræ, even when accompanied by considerable displacement, may be recovered from with a permanent unevenness of the spinous processes. When recovery is not reached, but the patient dies after many months' illness, a large quantity of new bone may be found, forming an incomplete shell around the broken part of the spine.

Particular interest attaches to the fractures *above the third cervical* vertebra, which cause instant death through stoppage of respiration, and which are sometimes mistaken for concussion of the brain, the spine having been forgotten in the post-mortem examination. Cases of fracture of the cervical vertebræ *below the third* generally live for two or three days, and then die from accident connected with imperfect respiration, or disorder of innervation of the sympathetic attended with a very high temperature. But in fractures in the *dorsal* region the sufferers generally live for several weeks, and then die either through the results of paralysis of the bladder, leading to cystitis and suppuration of the kidneys, or through bedsores and exhaustion, or pyæmia. Cases that end thus, after lingering many months with long-standing bedsores, may show lardaceous viscera complicating the other conditions.

But though simple fracture of the dorsal spine thus usually has a course of several weeks, yet in some cases the fracture is accompanied by fracture of the *sternum*; in other words, the skeleton is broken across the thorax. Such cases usually die more quickly. On the other hand, some remarkable cases are recorded of life prolonged for years after fracture of the cervical spine, especially one by Mr. Hilton, in which the patient lived fourteen years quite paralysed as to his extremities, and yet became so clever, by practising the use of his lips, that he could at last sketch very excellently. The *spinous processes* of the vertebræ are sometimes fractured without injury to the rest of the column.

*Fracture of the pelvis* is also a very perilous form of fracture. Its special interest and much of its danger arise from the relations of the *urethra* to the pelvic bones, which frequently cause the urethra to be ruptured in pelvic fracture. We know that the urethra pierces and is firmly fixed in the *triangular ligament*, and that this ligament is connected with the pubic arch very firmly, while the pubic arch is the weakest part of the pelvic bones. It hence arises that, when this arch is burst violently through—and pelvic fracture requires great violence—the triangular ligament is torn, and the rent implicates the urethra; for this soft mucous channel forms a large weak spot in the ligament that yields with especial ease. But, besides this way in which the urethra suffers, it may be crushed against the pelvic arch in falling astride a beam. In some very severe fractures we have known the pubic bones to be even forced back upon the sacrum so as to cut the prostate almost off the bladder, the bones recovering their usual relations afterwards through spasm of the muscles.

*Separation of epiphysis.*—This kind of accident can only occur in the young. The cartilage between the epiphysis and shaft of a bone forms a weak line in which a fracture might be expected to run. If this be borne in mind and looked for, it will be found to be a form of fracture not infrequent in children. Knowing that the growth of bone is effected by a continuous ossification of this epiphysial cartilage, it is natural to apprehend that the disruption of it will lead to a check in the growth of the bone to its full length; and some degree of shortening is ultimately found, though it is matter for surprise how nearly bones so injured do often come to perfect length and form.

We have already mentioned another peculiarity of fractures incidental to youth, in speaking of the bending-in of the cranium occasionally in young children. The same kind of accident will occur in their long bones, bending the bone instead of breaking it; of course, in such cases there must be a certain degree of interstitial crushing and disruption of texture. This condition has been called *greenstick fracture.*

*Spontaneous fracture.*—This occurrence generally signifies a diseased state of the bone, and often is due to *cancer* in the medullary cavity—a circumstance which will be indicated by the pre-existence of pain in the spot which has spontaneously broken. In such cases the disease of the bone, instead of producing a mass of cancer, may be found in the form of a cyst-like hollowing out of the shaft, containing a pulpy, gelatinous red matter, with the histological structure of sarcoma, or sometimes more closely resembling a localised patch of true osteomalacia.

But the bones may break spontaneously through mere *fatty degeneration*, though we cannot say that such an occurrence has come under our own notice. After thus breaking, it is remarkable that they may show a power of repair sufficient to heal the fracture, and it is still more surprising that, even in cases of cancerous fracture, repair has nearly succeeded in some instances. Lastly, in violent efforts perfectly healthy bones may be *spontaneously* broken, *i. e.* broken by the action of the muscles that naturally move them. This is especially the case with the patella and olecranon. Mr. Durham had a case of spontaneous fracture of the femur which perfectly united, and we have had a similar case of fracture of a rib which occurred during coughing. It has been shown by many observers that the bones of *lunatics* are soft or brittle. This is considered to be the reason why so many cases of broken ribs are observed in asylums. The explanation has been sought in the absence of reflex action, owing to disease in the centres; and therefore the bones are not prepared, as in health, for any undue blow or pressure.

**Rickets.**—The bones are, as we know, first represented by cartilage, which changes into bone by an interesting and curiously complicated process. This process, described shortly, consists of a multiplication of the cartilage cells, a calcification of the cartilage when rendered richly cellular through this multiplication, a formation of vacuolæ in this calcified material, and a production of true bone tissue within these vacuolæ. Although this process is so complex, yet, when all goes well, the area in which it is in course of proceeding is almost microscopically narrow. The completion follows the beginning of the change so quickly in the line of advancement that only a very thin layer of cartilage is found undergoing ossification at any period; and this layer forms properly a very even and local separation between the bone and the cartilage. Further, within this narrow advancing line the successive stages of the change are equally orderly, from the cartilage, through the before-mentioned modifications, layer by layer, to the bone beneath. When a part of the original cartilaginous shaft is converted to bone throughout all its thickness, consuming the cartilage until the bone reaches the periosteum, then the further growth of the bone in thickness is effected by the ossification of the deep layer of the periosteum. This is a much simpler process, which consists, shortly, in a simple calcification of the matrix, and transformation of the cells of the periosteum into bone-cells.

We can easily see that since the shafts of long bones are comparatively early converted to bone, while by their aftergrowth they become so large that the young bony shaft could be entirely lodged within the marrow canal of the adult shaft, it follows that, practically, the whole of the adult shaft is formed from periosteum. It may be remembered also that the bones of the vault of the skull are formed directly out of membrane without cartilage.

Now, the intermediate matter, which forms the stages of transformation of cartilage into bone, is of softer consistence than either cartilage or bone; and the same may be said of the substance which is intermediate between periosteum and bone; it is softer than either (hence the ease with which the periosteum may be stripped from the bone; such stripping is a tearing through of the deeper, softer, ossifying periosteum). From this it is easy to see why the amount of intermediate matter naturally present at any time should be small; for if a great quantity of it were present, the bones would be liable to bend or to break along the growing line, especially as the formation of the new bone on the outside is always accompanied by a removal of the older and deeper bone from the inside by the development of cancelli and of the medullary cavity. For as the shaft grows outward, the medullary canal is always increasing in the inside.

The disease known as *rickets* consists of a disturbance of the process of ossification, such as to delay its ultimate stages, while it hastens the earlier; thus producing a mass of imperfectly ossified matter upon the growing end of the young bony shaft; while it also softens the shaft itself by making a quantity of incomplete and weak bone under the periosteum. Meantime the medullary canal, widening, removes much or all of the better bone formed before the rickets set in. Thus the bone becomes thick, soft, and weak, its ends being swollen through the presence there of the actively growing cellular mass of disorderly ossifying cartilage.

Generally the epiphyses of the long bones are much enlarged, and the ends of the ribs beaded. It is in the sections of these bones especially that the peculiarities are seen. In a healthy bone the cartilage appears as a thin clear transparent blue line at its junction with the bone. In rickets this bluish line is much exaggerated, irregular in outline, and under the microscope shows as before said the cartilage not arranged in its usual order of rows of cells, but placed together in a disorderly manner. There is no actual disease of the bone, but a perverted process of growth, and this renders the bone soft, and causes it to give way from pressure.

The microscope shows the disease to consist of an intricate confusion of the order of the complex ossifying process, without any very wide departure from its essential nature. The multiplication of the cartilage cells progresses to an excessive extent. The calcification which should immediately succeed more or less fails. Yet the formation of medullary vacuoles occurs in the unossified mass, and, meanwhile, some of the descendant cells of the cartilage undergo interesting changes approximating their character to that of bone-cells, the interior acquiring a stellate figure resembling that of a lacuna. Thus we find, mingled together in chaotic confusion, crowds of multiplied cartilage cells, medullary spaces, and calcified patches, with various forms and degrees of transformation from the cartilage to the bone-cell.

These, heaped up, make the soft and swollen mass at the end of the shaft between the bone and cartilage. This soft mass is more vascular than normal, being permeated by numerous loops of vessels which run on often some distance into the cartilage itself. So, too, the periosteal layer of bone is thick, soft, and vascular, and the whole bone is everywhere congested, the contents of its medullary cavities being unnaturally red, while the fat disappears from them. In severe cases the connective tissue around the bone and among the adjoining muscles may take part in the process and undergo more or less swelling, so that the disease comes to wear some of the characters of an inflammation.

Much constitutional disturbance attends these more severe degrees of rickets ; so that the child may die with broncho-pneumonia, wasting diarrhœa, &c., if the case be acute or grave. But when the process is of medium severity, the local effects are those that chiefly command attention. The softened bones yield under the action of the muscles or the weight of the body, so that they become curved in bad cases to a surprising extent. This is more especially seen in the lower extremities. The femur bends at first outwards and forwards, and then again inwards, to meet its fellow at the knee ; the tibia and fibula in such cases bend outwards. In milder cases the tibia bends forward at its lower third. Sometimes the whole of the limb is bowed outwards. The bones of the arm are also curved, and sometimes a marked angle exists at upper part of humerus and lower part of radius and ulna. The process of growth is also inefficient, so that they become shortened, and thus the stature is stunted. The more pronounced curvatures are of great importance, especially those of the pelvis and ribs. These we shall describe when speaking of alterations of form in the skeleton. The curves will be observed in milder cases to be exaggerations of the natural slighter curvatures present in the bones, so that little or no deformity then results.

The process of rickets belongs only to the stages of ossification, passing off long before their completion. As the rickety condition disappears the bones undergo changes of an opposite kind. The thickened soft spongy substance becomes filled up with hard bony matter until the shaft is made very dense and solid, but permanently deformed by the curvatures that were caused during the active stages of the disease.

**Mollities Ossium, or Osteomalacia.**—This differs from the disease last mentioned in being one which affects the adult, and in most instances has come on in women during pregnancy; this, however, is by no means always the case, as the disease occurs in men, and in persons of both sexes, at advanced periods of life. There is, as in rickets, a preponderance of the animal over the earthy constituents, the latter gradually being lost, and having been observed in many cases to pass off by the urine; but, unlike rickets, the disease does not generally stop, but progresses in most cases to a fatal termination, during which time the patient is bedridden, and the different parts of the body have a tendency to fall together, the body becoming bent like a bow, and the limbs drawn together, and capable of being bent in all directions. The bones are so soft that they may be cut with a knife, and so light that they float on water. The earthy matter is all but gone, and nothing but the gelatinous part remains. In one of our specimens the bodies of the vertebræ have so shrunken that the intervertebral cartilages almost meet.

The disease shows itself first in those bones which have but little compact substance, such as the pelvis, vertebræ, ribs, &c., and later in the hard shafts of the femur, humerus, &c. In all cases it tends to spread and involve the whole skeleton.

To the naked eye the disease appears as a widening of the medullary spaces at the expense of the bony tissue, the contents of the spaces becoming red and gelatinous, while the trabeculæ and plates of the spongy bone grow thin and soften, until many disappear, losing their lime salts first, so that they can be cut with a knife, or bent like horn. The microscope does not reveal much in explanation of the process; we find the bone-corpuscles altered in form, losing their fineness of outline and regularity of appearance, so that they become widened and irregular in shape, and appear to have fewer canalicular processes; meanwhile, the medulla is congested intensely, and is found to contain an excessive proportion of lymphoid corpuscles such as are naturally present in smaller number, while its fat generally disappears. But at what appears to be a later stage some cases show less of congestion, the cancelli containing a white gelatinous or fatty degenerate matter.

The specific gravity of a vertebra in the Museum, taken from a case of osteomalacia, is scarcely ·7, while healthy bone is nearly two or three times as heavy. The bones may bend and subsequently unite, as seen in the ribs. The scapula may become thin as paper, and doubled upon itself, so also may the os ilii and sacrum.

*Other forms of softening.*—Although the condition of bones understood by rickets is pretty well defined, as is that in the adult known as osteomalacia, yet we sometimes meet with cases which can scarcely fall into either category. Thus there have been two cases of late years in the hospital where the bones of infants had undergone a remarkable softening, and yet did not put on the appearance significant of rickets. In one of these cases examined by Dr. Goodhart he was at a loss to know whether to style it infantile osteomalacia, or be content with the word softening and overgrowth of bone. In this infant, a little more than a year old, the bones and epiphyses felt enlarged, and when examined after death were seen to be enlarged, granular, and easily cut with a knife. Not only were the long bones so affected, but the bones of the cranium were large and spongy. This enlargement and rarefaction of the bones reminded one of the state of the skeleton in osteitis deformans. Mr. Bland Sutton had observed a similar softening of bones in caged monkeys, but this condition had been usually styled rickets, and was attributed to malnutrition from improper food.

Mr. Lucas has described cases of softening of the bones in boys about the age of puberty, beginning with an arching of the back and bending of the limbs. In many of these cases there was albuminuria, and Mr. Lucas was inclined to give to the complaint the name of *late rickets.* Mr. Davies-Colley had the case of a lad in which he felt doubtful whether to adopt this name or osteomalacia, asking himself meanwhile if there were any real difference between them. He found the periosteum thick, the medulla very vascular, the bones soft, parts of them having a gelatinous appearance, being composed of a homogeneous matrix of a soft material. The long bones showed very little true osseous substance in the shape of Haversian canals, but were composed of a porous or trabecular material. The bones of the cranium were also porous and thick. Mr. Davies-Colley conceived that the state of skeleton might have been due to some general inflammatory process, causing absorption of the previously compact tissue.

**Alterations in Form of Head, Spine, Thorax, and Pelvis.**—Instead of speaking of the changes which take place in the various bones under the different forms of maladies which cause them, it will be more convenient to review the deformities of the particular parts of the skeleton at the same time, so as to compare them together.

*Head.*—The most remarkable alterations of the skull are produced by *hydrocephalus.* In this disease the fluid in the ventricles of the brain expands the head, and the bones of the calvaria are widely spread and separated. Such skulls offer several characteristic peculiarities. There is, first, a great disproportion between the enormous size of the cranium and the comparatively very diminutive facial bones below it. The vault is very thin, and for a long time is in part membranous, and the indentation of the sutures is deep. The supra-orbital ridge is done away with from the stretching upwards and forwards of the frontal bone. If the child live and the fluid is not absorbed, fresh points of ossification arise, and thus supplementary bones grow in the interstices of the natural ones. These are of various shapes and sizes, and are named ossa triquetra, or Wormian bones. We see such bones of very great size in the remarkable hydrocephalic head of the man Cardinal, which measures thirty-three inches in circumference and held a gallon of fluid. The form of the orbit is very remarkable in hydrocephalic crania; the roof of it, instead of being horizontal, is inclined more to be vertical, and this gives the eyes the peculiar roll so characteristic of hydrocephalus; at least, the peculiarity of the orbits is found in all the infants' skulls in our Museum, though it lessens in those who live to be older.

In *rickets* also the cranium is disproportionately large and often overhanging in front, but a rickety cranium does not afford such evident characteristics as those which belong to hydrocephalus. The sutures and fontanelles are late in closing, and during the progress of the rickets, which may be at any time from birth to the tenth year, the bones are light and thin and porous, being hyperæmic when fresh. They may also show gaps in their continuity along the course of the sutures, especially the lambdoidal, these gaps being filled in with membrane; but afterwards, when the disease is healed, the opposite condition establishes itself, and the bones are found dense and thickened along their edges, while their middle parts may be either thin and hard, or natural, or perhaps very thick; but it is still a question whether the great extent of porous thickening of the cranium we have before described is due to rickets or not. It is certainly sometimes associated with it, while it is equally certain that great rickety deformity of limb may be found with a normal cranium, the large size of the head being only relative.

The skull is often found remarkably thinned in infants, or the bone altogether wanting in places, a condition which has been styled *craniotabes.* This has been described as occurring in rickety children, but Drs. Barlow and Lees by collecting a large number of cases found that a history of syphilis largely prevailed, so that they felt no doubt that this disease lay at the foundation of the atrophy. During life the skull feels in places, and more especially over the parietal and occipital bones, like parchment, or pressing on a bladder. On examination after death nothing is found but membranous tissue. In our Museum are remarkable examples of crania where the bone is deficient in places, these being occupied by membrane, and others where they correspond to the sulci of the brain. There is no proof that these membranous opercula were due to syphilis. Parrot thought that pressure had much to do with the thinning of the bone; the combined pressure arising from the frequent presence of fluid within and from the weight of the head without in lying down.

Our Museum also presents some other curious specimens of malformed and unsymmetrical skulls. In one will be seen the sides flattened and raised up in ridges towards the top; in another the sides are not alike; and there is a skull of the flat-headed Indian, the front part of whose head was thus flattened in infancy by artificial pressure.

*Spine.—Angular curvature, i. e.* the bending of the spine to an angle projecting backwards, is called *kyphosis.* It results from disease and destruction of the bodies of the vertebræ and their consequent falling together; this occurs mostly in the dorsal region; indeed, caries of the lumbar and cervical spine do not often produce this curvature.

Where the bodies of several dorsal vertebræ are destroyed the curvature of the back is excessive, as in a case where the nine dorsal and three upper lumbar are excavated away, and their remains form a small angular mass, to which the transverse processes and ribs are attached. In some cases the remains of the vertebræ are held together by ligamentous tissue. The cord is bent and follows the inequalities of the spine; and it is remarkable to what extent the bones may be affected without damage to the cord.

*Lateral curvature.*—When the spine is bent in a curve to one side, the term *skoliosis* is used. A slight curvature of this kind is very common, especially in women; but there seems no limit to its extent, until, indeed, the spine shall be bent down parallel with itself, as is seen in a very remarkable specimen, where the ribs actually pass down in front of it. Skoliosis does not result from caries of the spine like angular curvature. Its cause is in fact comparatively obscure. Lesser degrees of it are found in rickets, but the more severe examples are found in persons who do not show any of the characteristic results of rickets, such as peculiar bends of the long bones, enlarged epiphyses, &c. It will be seen, on examining our specimens of lateral curvature, that the vertebræ themselves are not much altered in shape, but the bend is due almost entirely to the wedge-like form of the cartilage between them, this being wide on the convex and narrow on the concave side; on the latter the bones actually touch, and in some cases are superficially ankylosed, and so likewise in the concavity of the bend the heads of the ribs are so crowded together that ankylosis may take place both with the vertebræ and with one another. Mr. Lane thinks that these cases arise from pressure, and vary in form according to the employment of the individual. Thus a simple curve in the back is not uncommon. By regarding attentively a spine thus laterally curved we see that the vertebræ have suffered a rotation on their axis, or have so turned on themselves that the bodies of the vertebræ form the prominence of the convexity, as will naturally be the case if a spine be forcibly bent downwards, while the spinous processes are turned to the concavity, one set of transverse processes projecting backward. One side of the chest is necessarily carried backwards in this rotation of the spine, and thus the hump on the back is formed by the angles of the ribs as well as the transverse processes of vertebræ.

In some cases, as in rickets, the spine is so curved that the lumbar vertebræ, with the promontory of the sacrum, project much forwards, and the back necessarily falls in. This curvature is called *lordosis.*

In *mollities ossium* the tendency is for the head and pelvis to fall together, and the spine thus makes a long curve backwards in the opposite direction to that in lordosis.

*Thorax.*—In lateral curvature the thorax is necessarily much distorted, one side being narrowed and the other widened. By looking at our preparations we see what generally occurs. The spine is here bent so that the convexity is on the right and the concavity on the left; as we have already seen, the ribs on the convex side are carried backwards by the rotation of the spine, which always makes the bodies form the front of the curve. Thus the ribs and transverse processes together form the prominence of the hump-back. The ribs so carried back have necessarily to run a longer forward course to the sternum, which retains its natural position. To do this they are stretched out straightly, so as to make the antero-posterior diameter of this side of the chest great and its lateral diameter small. On the other hand, the ribs on the concave side, being necessarily carried forwards by the twist of the spine, have a correspondingly shorter distance to run to the sternum, so that they dispose their length in a stronger lateral curve, and thus widen out that side of the chest, while its vertical depth is, of course, smaller than on the other side. Thus it comes about that in skoliosis the lateral measurement of the concave side of the chest is great and the antero-posterior and vertical measurement small, while, on the convex side, the lateral measurement is small and the antero-posterior and vertical great. The ribs on the concave side may meet each other and become ankylosed, especially near the spine. The sternum keeps its natural position in the median line. In slighter lateral curvature the chest may be bent a little to one side with the spine, without much alteration of form. On the other hand, when there is a double curvature the alterations may be so complex that it is scarcely possible to describe them. The usual condition in lateral curvature is, however, such as we have stated above. In *angular curvature* or *kyphosis* the chest is symmetrical, but bends down towards the pelvis, so that the lower ribs reach below the ilium. The vertical measurement is decreased, while the antero-posterior is considerably increased and the sternum is carried forward, so that it is in a vertical plane considerably anterior to that of the face.

In *rickets* the thorax presents a characteristic kind of alteration, corresponding to what is popularly called "pigeon breast," a name given to it from the prominence of the sternum. In this disorder, as we have already said, the bones are soft, especially the growing ends of the bones. At the point of junction of the rib and cartilage the row of beads can be felt as before mentioned. After death they may be very noticeable on the inside. Owing to the softening the ribs do not firmly and equally resist the atmospheric pressure in the act of respiration, as they normally should, but yield, so that the softer parts recede. Thus it comes to pass that the line along the thorax, which corresponds to the softened ends of the ribs, sinks in, forming a groove on each side, giving relative prominence to the sternum. This groove on the left side is shallower on account of the presence of the heart beneath. On the right side it is deeper until we reach the lower part, where the liver lies beneath supporting the ribs. Besides this lateral groove there is a transverse one below the xiphoid cartilage. Of course this effect of atmospheric pressure will be more evident if the entry of air into the chest is not free, since then the breathing efforts will be more forcible and the suction greater. Indeed, in some cases, a similar "pigeon-breasted" state of chest may come about without any evidence of rickets being produced through any of those causes, such as enlarged tonsils, small trachea, capillary bronchitis, &c., which permanently obstruct the entry of air.

On the other hand, when the original expansion of the lung substance is imperfect, so that a part of it remains airless in the condition called *atelectasis*, or when through *whooping-cough*, &c., parts of the lower lobes are shrunken permanently, so as to diminish the size of the lower parts of the lung, the lower part of the chest is shrunken in comparison with the upper part. This contrast between a large upper and a small lower part of the chest is increased by the occurrence of compensating expansion of the upper part of the lungs such as we shall describe.

In *mollities ossium* the parts of the chest have a tendency to fall together, the ribs are softened, some fractured and bent into various shapes, and the chest falls in at the sides. The spine is curved, forming a convexity backwards; and the sternum is often bent in its middle.

In *emphysema* the thorax is permanently in a condition of extreme inspiration; indeed, more expanded than in ordinary inspiration in a healthy chest. The lungs having lost their elasticity, the act of inspiration is only effected by muscular effort and the recoil of the ribs —processes which naturally constitute only the earlier, if the greater part, of the expiratory act. This act is never finished as it should be by the elastic recoil of the lung, and fresh breaths are taken into the lungs already partly full, until the muscular effort required to renew the air is so great as to largely increase the curvature of the dorsal spine. For the inspiratory muscles draw down the upper part of the spine in raising the ribs, and the expiratory muscles draw up the lower part of the spine in depressing the ribs, and the spine above is drawn downwards by the first and upwards by the second, so that in this way the dorsal part of it becomes bowed backwards in the way so characteristic of old asthmatic or emphysematous subjects, thus increasing the antero-posterior diameter of the chest. Such subjects are also high shouldered, for, of course, the shoulders go up with the chest. Other changes may occur in the chest from disease. Thus, from effects of pleurisy, one side may be contracted, and even the spine be bent; and, from the same cause, a local contraction or depression may arise at the lower part of the chest, or at the upper part beneath the clavicle.

*Pelvis.*—This may be variously misshapen. First we shall consider the effects of rickets and osteomalacia. The respective effects of these on the pelvis differ almost characteristically. In *rickets* the sacrum and lower lumbar vertebræ are bent forward (lordosis), so that the former approaches and almost meets the symphysis pubis, which is pushed upwards to meet it. The cavity of the pelvis is thus narrowed from before backwards, especially in the middle, while it is elongated from side to side, thus, on the whole, resembling somewhat the figure of an hour-glass. It is not difficult to understand how this shape is produced if we recollect that the growing edges of the bones are the soft parts in rickets, and that the growing edges are in the acetabulum, the sacro-iliac synchondroses, and the symphysis pubis. Those being the soft parts, and the thrust being made from the spine to the thigh-bones, so as to squeeze the pelvis between them, we see that the femoro-vertebral diameter will become narrow, the softish acetabula giving way towards the spine, thus flattening the pelvis from before backwards and producing the hour-glass shape when the symphysis goes with them; but in some cases of rickets the hour-glass shape is not so evident, for the symphysis pubis projects forwards, making the inlet of the cavity triangular. Dr. Champneys has thrown considerable doubt on this explanation, for he asserts that the upward pressure of the femur would not force the rim inwards, but rather tend to evert it. In *osteomalacia*, or mollities ossium, the softness of the bones is equal in all parts, and hence not only is the sacrum bent downwards, but also in bad cases the bone about the acetabulum on each side is thrust upwards towards the sacrum, leaving the symphysis pubis projecting like a beak. The bones, even the alæ ilii, bend freely whilst yielding, thus producing an appearance which is very surprising in comparison with the usually hard and incompressible state of the pelvis. On the other hand, in the rickety pelvis, though the front of the pelvis with the pubis is pushed backwards towards the spine, yet there is no bending of the other pelvic bones.

In *angular curvature* of the spine the pelvis is generally deep, like the thorax, as may be seen in our specimen.

In *lateral curvature* the pelvis generally becomes slightly distorted, owing to its sinking down on the side of the curvature. In this specimen it will be seen that on the side of the convexity the pelvis is raised, so that the tuberosity of the ischium on this side is higher than that on the other, and the pelvis is necessarily distorted.

The *oblique pelvis* of *Naegelé* is due to ankylosis of one or other of the sacro-iliac synchondroses occurring in infancy, or even in intrauterine life, so that during development a distortion naturally ensues. We may look upon the pelvis in our Museum as a specimen of the affection. We see the right sacro-iliac joint ankylosed, and the pelvis is unsymmetrical. The disease of the joint has been accompanied by much loss of substance to the ilium and sacrum on the right side, and thus the latter bone is thrown in this direction, and the whole right side of the pelvis is smaller than the left. Hence the front of the sacrum does not face the symphysis pubis, and if the diagonal measurement be taken from this diseased joint to the left acetabulum on the opposite side, it will be seen to be greater than that taken in the other diagonal.

It is said by some that the deformity occurs without disease of the joint, and that the sacrum may be found wasted from birth ; or any unequal pressure may throw the pelvis on one side.

*Spondylolisthesis* is the case where the pelvis is narrowed by the slipping forward of the last lumbar vertebra on to the sacrum. It may be found projecting forward so as to compress the uterus between the pubes and bodies of vertebræ. There is a marked depression or lordosis of the lumbar region. We shall allude to it again in the next section.

**Morbid Growths.**—The tumours that form from bones present a general resemblance to the tumours that form in the soft parts. But in accordance with a rule which we shall frequently have to mention, by which tumours have more or less of the structural character of the tissue they form in, the tumours of bone are more or less like bone, the resemblance being generally greater in proportion to the slowness of their growth. Such resemblance was thought to be due to some obscure influence the surrounding tissue had on the development of the newly formed material. But there is great reason to think that the new material of the tumour is derived from the bone by a direct multiplication of some of its structural elements ; so that the likeness of the tumour to the matrix, or part it grows in, is an hereditary likeness due to descent. But, as we have before had occasion frequently to remark, a whole bone consists of several tissues, each distinct in character from the others. Thus there are at least these : cartilage, periosteum, medulla, tela ossea, and vessels.

A new formation from bone may take its rise in, and its characters from, either of these components ; so as, in short, to be cartilaginous, or fibrous, or myeloid, or osteoid, or vascular. When the growth is slow, the character of one or other of these tissues is tolerably well observed in the tumour ; and then generally the tumour is "innocent," or devoid of those infectious properties that constitute malignancy. But, if the growth is rapid, the new formation does not acquire the special characters of the tissue it arises in. Its elements, hastily produced, show much of their original round unspecialised shape, or have within them, in the form of large nuclei and nucleoli, evidences of their vivid reproductive activity.

Besides their liability to primary tumours, which is relatively considerable, the bones are also not infrequently the seat of secondary cancer, which, indeed, may be widely distributed in the skeleton, as we shall see. The tumours which most commonly arise in bone are—

*Osteoma.*—This name is given to such tumours as are *practically* composed of bone. When they grow from the surface of a bone they are called *Exostoses;* when they form in the interior of a bone they are called *Enostoses.* The relations of exostoses to inflammatory nodes, &c., on bones have already been considered. We must remember that the *tela ossea,* or proper hard substance of bone, is mingled always with certain soft tissues, from which it arises and by which it is supported. It will be readily anticipated that pathological new bone, formed as it is under abnormal conditions, and without the harmonising influence of natural development, will often show a disproportion of the several constructive elements found in healthy bone. Thus we sometimes find in bony tumours an excessive development of the *tela ossea,* so that the bone becomes as hard as ivory, and almost entirely devoid of medulla, and even of vessels; such tumours are called *Ivory exostoses.* Their favourite seat is about the face and anterior part of the skull near the face, especially near the frontal and other sinuses, where they form more or less rounded nodulated tumours of extreme hardness. The ivory change appears sometimes to go on until it destroys the life of the tumour by stopping circulation in it; and so in a remarkable case, described by Mr. Hilton in the 'Guy's Hospital Reports,' the tumour may spontaneously separate. This form of tumour has often been observed to arise within the diploë of the skull in the frontal region, growing, and separating the tables from each other, until it bursts through one or both of them. Such an occurrence is seen in a case of Mr. Birkett's, which is also described in the 'Guy's Hospital Reports.' It is for such a production of bone that the term enostosis is used.

Other exostoses show natural cancellous tissue, and occasionally one arising from a long bone contains a marrow canal in communication with that of the shaft of the bone.

These bony tumours do not only vary in the relative density or rarity of the bone that composes them, but they also offer a considerable variety in the amount of the associated soft tissues—cartilage, periosteum, medulla, &c.—which accompanies them in a more or less modified state. Thus many bony outgrowths have a covering of cartilage, which forms their growing part, and from which the bone develops in the growth of the tumour. In such cases it is not easy to decide whether to call the tumour an ossifying enchondroma or a cartilaginous exostosis; but this difficulty is only verbal. It is practically settled by calling the tumour after the constituent that enters most largely into its composition. On the femur such tumours are not infrequent. Facts have been adduced to show that such cartilaginous exostoses affect most frequently the situation of the epiphysial cartilage, as though they took their rise from that. But when we remember that, in the reproduction of bone after fracture, or in the production of bone by transplanted periosteum, a cartilaginous stage is often passed through, so that periosteum can thus be made to change itself into cartilage, there appears no occasion to go far to search for a reason why exostoses should be tipped with cartilage. Such an exostosis, further, may have a bursa over it, and the sac of this bursa may communicate with the cavity of a joint. When exostoses grow near a great joint, this is a possibility that must be carefully weighed by the surgeon before operating. In yet other cases the new growth of bone occurs within a great mass of a tissue more or less resembling the periosteum, and this tissue sometimes is more like fibrous tissue, and at other times more resembles cartilage. In such cases it is often quite a question of words whether the tumour should be called an osteoma or an ossifying fibroma, when the ossifying tissue is fibre; or an osteoid chondroma, when it has a structure resembling fibro-cartilage. But in the latter case the tumour is of a more doubtful and suspicious character, so that it is more important to make the distinction; many very large, dense, bony tumours are of the nature of osteoid chondroma.

In short, there are not many osteomata or tumours of pure bone above the dimensions of exostoses or mere bony outgrowths. The greater tumours, which contain a large proportion of bone, show along with it an important amount of some one or other of the allied tissues, such as periosteum, cartilage, medulla, &c., whose subsequent and final changes end in the increase of the bony mass by continuous transformation of their tissue.

*Enchondroma.*—The bones are the favourite seat of enchondroma, and those enchondromata which occur in bone are generally more purely cartilaginous in their structure and more innocent in their tendency than the rarer enchondromata which arise in the soft parts; for instance, in the testis. Virchow has advanced the belief that tumours which in structure resemble the part they grow in are generally of an innocent nature; and remembering that cartilage is nearly always united with every bone, and forms a stage in the development of most bones, its presence in bone is not a great departure from the nature of the parent tissue; so that thus, on Virchow's view, an enchondroma in a bone comes near to possessing that local homology which he regards as the criterion of innocence in tumours. On the older and simpler view of homology which considered as homologous and innocent any tumour whose substance was like any texture of the body, wherever it might grow, the cartilage tumours of bone are the very purest type of homology and innocence. Their histological structure departs very little indeed from that of ordinary permanent cartilage.

But the usual seat of an enchondroma in a bone is where cartilage is not naturally present, as within the shafts of the long bones and phalanges, or on the surface of the flat bones. Virchow suggests that an included portion of unossified cartilage remaining from former rickets may explain this, but no facts are adduced to show the actual truth of this view. The bones of the limbs are liable to cartilaginous tumours in proportion to their distance from the trunk. The tumours are often multiple; thus they may affect many bones of the hands, and produce enormous deformity. They are observed to arise either within the bone or on the surface, and have been divided accordingly into *peripheral* and *central* enchondromata. Those of the long and short bones generally arise centrally, and those of the flat bones, as the pelvis and scapula, usually arise peripherally. In the former case they expand the shaft of the bone to a mere shell, which, spread over them, holds their surface into some smoothness and evenness, while in the latter case the tumours project freely and have a more uneven surface, from the separate freedom of the lobes of the tumour. Enchondromata are of a lobulated structure throughout, so that, on section, one sees the clearer bluish cartilage divided into areolæ by plates and beams of a more fibrous whiter substance, into which vessels extend. The cause of this lobulated structure appears to be the increase of the tumour by the starting up of fresh centres of growth in the tissue on the borders of the main mass, which then grow and coalesce with it. Where one of these enchondromata is irremovable from its position, so that it grows very large, as in the scapula or pelvis, it is apt to soften in parts to an almost gelatinous consistency, or even in places to melt down as it were, and form cysts. It is then called *cysto-enchondroma.* Some enchondromata have cysts that are not explicable in this way. The increase of the tumour may stretch the teguments until they ulcerate, and some portion of the tumour may break down and be discharged; producing, from the size of the mass, and the great excavation in it, a frightful degree of misery.

In one case of Mr. Birkett's, which we inspected, the tumour found in the scapula weighed many pounds. Besides the softenings they undergo, enchondromata vary in their consistence. It is generally admitted that those which are softer are of less benignant disposition. Their microscopic structure then approaches that of myxomata, and they are called *myxo-enchondroma.* Some of the softer enchondromata have been known to recur on removal, as in the remarkable case from which one of our preparations was taken. The tumour occupied the upper jaw, and after growing nine years was extirpated by Mr. Morgan; it sprouted again, so that at the patient's death, seven years after the operation, it had reached the immense size seen in our cast. Other examples of enchondroma have proved infectious to remote parts. Thus an enchondroma of the scapula has been associated with similar disease in the lung.

We may here mention the occurrence of small outgrowths of cartilage called *ecchondroses* from the permanent cartilages, such as those of the ribs, ears, symphyses, &c. In these situations they have not reached any important size; but in the larynx they have been known to project as polypoid growths from the cartilages. Such ecchondroses of the articular cartilage may take part in diseases of the joints, and will be mentioned in that connection.

We have several times met with a soft mucoid mass under the pons Varolii, between it and the basilar bone. It is composed of delicate round cells. In the cases we have seen the growth did not appear to have exercised any pressure; it was evidently the same thing as was by H. Müller referred to a persistence of the top of the fœtal notochord, and which Virchow has proposed to call *chordoma.*

*Osteoid chondroma* (*osteoid cancer*).—This name, which we have already several times used in connection with fibroma and osteoma, is one that very well expresses the structure of the soft growing part of an important kind of rapidly ossifying tumour, which often grows to a large size, and which also has a serious interest from the tendency which it undoubtedly has to malignancy. So many recurrent ossific tumours have been recently found to be of this kind that it is doubtful whether most of the so-called "osteoid cancers" of authors were not really osteoid chondromata. They form dense, hard, heavy tumours, a large part of which is nearly always composed of bone springing from the periosteum, and also extending inwards to fill up the medullary canal, thus obliterating the outline of the original bony shaft. They grow very frequently on the articular end of a long bone, especially about the knee-joint. The tumours they form are tolerably uniform in outline, but have lobes of low elevation, separated by grooves in which vessels, nerves, &c., are lodged. The structure is a dense whitish fibrous tissue, grating on section, and having a close tendon-like texture. The microscope shows a structure which can best be compared to those plates of osteoid cartilage which are so common on the spinal pia mater after middle life, *i. e.* it closely resembles bone which has been decalcified by acids, and its transformation into bone is simply effected by the addition of lime salts to the matrix between the cells, which themselves become bone-corpuscles. We once had in the hospital a man with a very large tumour of this kind at the upper end of the humerus. In the course of his disease painful paraplegia came on, and after death there was found an exquisite example of the same osteoid chondroma in the middle dorsal vertebræ, and a great number of similar tumours were present in the lungs. It is this tendency to metastasis which earned for it the old name osteoid cancer. The name osteoid chondroma is preferable, as it refers to the recent and active part of the tumour in which its dangerous activity lies, for when once the mass has ossified it remains stationary, the morbid bone being, so far, as innocent as any natural part of the skeleton. The mischief runs on through the production of the new-formed cartilage in the new exterior advancing part of the growth. Attention must always be mainly directed to the active growing parts of tumours.

*Fibroma.*—Fibrous tumours are not often found arising from bones; most examples recorded or preserved in Museums have affected the jaws. Some have grown within the bone and expanded it. Others arise from the periosteal surface, especially that of the alveolar border of the jaw, so that the tumour in its growth raises the gums, whose tissue is spread over it, thus making one sort of *Epulis*, for by this name any tumours of the gums are called. Such fibrous tumours are generally permeated by a more or less dense framework of bone, which extends from the affected part of the jaw, and spreads in dendritic form through the growth continually increasing by ossification of the fibrous tissue (osteo-fibroma). Pure fibromata are very rarely found anywhere else on the skeleton. We have one specimen on the phalanx of a finger, and one has been occasionally met with in other parts. But although tumours of pure fibrous tissue are thus rare, yet fibrous tissue is found not infrequently forming considerable parts of mixed tumours, such as *fibro-enchondroma* or *fibro-sarcoma*, wherein strong and broad bands of fibrous tissue pass through the tumour, dividing it into lobes, and composing a greater or smaller part of it. That fibrous substance, which is present in many large growths from bone which are called ostèoma from the prevalence of bone in them, is often mistaken for mere fibrous tissue, when closer examination will show it to have the structure we have just described as osteoid chondroma.

A tumour is sometimes met with in the lower jaw composed of dentine. It is excessively hard, and the section has a radiating appearance. Mr. Tomes calls it "labyrinthine dentine," and has named the tumour "*dentoma.*"

*Sarcoma.*—When considering sarcoma in comparison with fibroma, osteoma, enchondroma, &c., we observed that sarcoma shows this important difference from those classes of tumours, that there is no standard normal tissue to which we can refer its structure for comparison. There is no normal complete tissue like in structure to sarcoma.

The essential feature of the histology of sarcoma is the presence of cells in an intercellular tissue, forming a homogeneous web. The cells are found to vary very much. Thus, some are spindle-shaped, large or small, others are round cells, large or small; and, besides, there are found in some sarcomas cells with many nuclei; they are very large, and generally indented at their edges by the pressure of their neighbours. These are known as giant cells from their size, or myeloid cells from the presence of similar cells in fœtal marrow.

The sarcomas of bone are conveniently divided into those which arise externally and those which arise internally, or *periosteal* and *endosteal.* This division does not separate all of them clearly into two groups, since it is often difficult to say whether the tumour arises from periosteum or medulla; yet it constitutes a convenient division, for, speaking generally, the periosteal and endosteal sarcomas show characteristic differences each from the other.

The *periosteal sarcomas* form tumours whose consistence varies from a fibrous toughness to a fleshy or even tremulous softness, and they are, as usual, soft in proportion to the rapidity of their growth; they have an elastic consistence, and do not yield a milky juice when scraped. The surface of the tumour is generally in bold convexities, either incapsulated in fibrous tissue, which it is unable to infect, or if it be more diffusive and infectious, extending into the muscles, &c., which it is meantime pushing before it in its growth. Such a tumour is very liable to ossification of its structure. The new substance takes its rise close to the bone under the periosteum in the deep layer of that membrane which Müller called its "cambium layer." In its growth it elevates the periosteum off the bone, and in some instances the periosteum of the marginal parts of the tumour thus displaced undergoes ossification in some points of its new position, forming a layer or layers of bone at an angle with the shaft. When it has grown large such a tumour will nearly always afford a considerable variety of appearances in different parts. Blood will often be effused in some places, and when found in various stages of decay will offer different shades, from red, through brown, to pale yellow cheesy colour; or cysts will be present in the tumour, arising as apoplectic cysts from hæmorrhage, or else from softening down of the tissue to form cavities, or sometimes by active development of cystic spaces lined by epithelium. The tissue of the growth itself may often differ in different parts, some parts having a somewhat mucous or glutinous consistence from the presence of some mucin in the intercellular substance (myxosarcoma), while others are tough and fibrous, showing a quantity of pure fibrous tissue in their composition (fibro-sarcoma), in others yet more or less extensive patches of cartilage are found (chondro-sarcoma), while in others, and this frequently, there is a large development of bone ramifying through the mass (osteo-sarcoma). The bundles of fibres and cells in the mass wind about irregularly, and the section dividing them at different angles will cause a faintly mottled appearance, while, lastly, the fatty decay of portions of the growth often produces more obvious opaque mottlings. All these appearances may be found in a single tumour; more commonly they are not all present, but there is a prevalence of one of the varieties of composition described, and from this prevalence of or purity of one variety, the tumour would be called fibro-sarcoma, myxo-sarcoma, chondro-sarcoma, osteo-sarcoma, cysto-sarcoma, hæmorrhagic sarcoma.

The interior of the bone at the affected part may undergo simple induration only, the excess of supply in the close neighbourhood of the tumour inducing excessive growth; or else, and in many cases indeed, the tissue of the tumour is found not only outside, but also in the interior of the bone.

Sarcomatous tumours are found more frequently near the articular ends of the long bones than elsewhere in the skeleton. They do not implicate the joints, at least only in very rare instances, which we never met with ourselves. A remarkable case is, however, given by Dr. Steudener, of Halle, where a round-cell sarcoma, reaching the knee-joint, grew freely in the synovial membrane, and in that only. They are sometimes met with in the upper jaw, and consist of both the round-celled and spindle-celled variety.

The bony development in osteo-sarcoma may reach a curious degree of complexity, forming, when prepared as a skeleton, a number of beautiful spicules, proceeding from all around the bone, and branching as they extend, to terminate in an even coral-like surface in the outer part of the growth. The bony skeleton of the tumour is generally stout and firm, in proportion to the firmness of the tumour, and that is proportioned to the slowness of its growth, as we have, indeed, already said.

At first the periosteal sarcomas are kept in by the periosteum, but soon the rapidly growing examples find their way through this membrane and spread in the tissue outside, into and among the muscles generally, in bold, advancing, round masses of the flesh of the tumour, which may be seen partly destroying the muscle by coarse pressure, and partly transforming it by spreading minutely along the interstitial tissues between the fibres, and so crushing these out of existence.

The histological structure of the periosteal sarcoma shows in different instances, or perhaps in different places in the same instance, all varieties of the proper structure of sarcoma, that is, a homogeneous tissue of round or spindle-shaped cells, large or small, with more or less of an intercellular tissue, connecting them into a web. Sometimes there is a small proportion of giant cells, but not many, and doubt has been raised whether the examples with giant cells are not in reality developed from the medulla rather than the periosteum. Besides thus varying in the kind of sarcomatous tissue present, there are further and wider varieties of microscopic structure corresponding with those variations of the tumour into myxoma, enchondroma, osteoma, &c., which we have before mentioned; we might say that such varieties show all the microscopic characters of these several kinds of tumour, and all stages of intermediate growth between them.

Although the periosteal sarcomas are broadly distinguished from the sarcomas that arise in the medulla, yet, as we have already hinted, many periosteal growths show so much coexistent endosteal formation that it is difficult to decide whether the outside or inside of the bone is most affected. In a child aged four there was multiple sarcoma of the bones; the cranium, ribs, sternum, &c., being affected. On the cranium the tumours were still covered with periosteum, but within they had penetrated the dura mater. They contained round and oval nuclei, and might be called round-celled sarcoma. No other structures of the body were affected.

*Endosteal sarcomas, myeloid tumours.*—Those tumours which arise in the medullary tissue and distend the bone have always created more surprise than the kind we have just described. Indeed, when we find the shaft of a bone, rigid as it naturally is, widened so as to form a great bony sac, there is reason for wonder, and some observation and reflection are required to explain the change. Such expansion of the bone arises in this way. While the pressure of the growth within removes the old bone by absorption, it presses out the yielding periosteum, which, while being very gradually thrust into a new position, continues to make new bone as it goes, thus keeping a shell of new bone more or less continuous, when the process is sufficiently slow. But, on the other hand, if the process of expansion is very rapid, there is no time for such ossification, and the whole bone is softened away at that part by the growth, so as to seem to cease abruptly as it enters the tumour. When the substance of the tumour is removed from the interior of such a bony sac, as when the bone is put up in the form of a skeleton preparation, the effect is very remarkable, and all Museums contain some such specimens under the old name *spina ventosa.* The kind of endosteal sarcoma which thus distends the bone is in the form of a growth, often having a peculiar deep crimson-red colour, and being soft and dry, not at all fibrous, and yet not yielding any juice on scraping as cancers do. The red hue of the substance is very peculiar; it formerly gave the name *spleen-like* to the tumour; it is somewhat like plum juice, and is different from the colour of any natural tissue of the body. There is often some blood extravasated in the tumour, which may increase the redness; but when this is removed the substance itself is found to have this red hue. In some cases the tumour is soft throughout, in others processes of fibre proceed from the outer surface, and slit up into a reticulum more or less dense, constituting thus a *fibro-sarcoma;* in other cases cysts are formed in it. The most usual seat of such tumours is the lower or upper jaw, and the next most frequent seat is the end of a long bone. Beginning in the centre of the bone, the growth continues until the shell of bone is reached and lifted, as already described. The process in its spread encounters at last the articular cartilage, but this generally is not involved, and in our specimens, taken from young subjects, the epiphysis is not involved. The microscopic structure long ago arrested attention from the peculiarity it has in the great proportional quantity of very large "giant cells," which interestingly resemble the large multinucleated cells of fœtal marrow, suggesting a return of the fœtal type in the growth of the tumour. These bodies have been called *myeloid cells*, and the tumours themselves were hence called myeloid, when it was not sufficiently recognised that these large multinucleated cells are very often present in morbid growths, and are not limited to the tumours which occur in the medulla of bones, but are also found in myxoma, and in lymphoma, and even in tubercular formations. They are now known only as *giant cells.* However, in the "myeloid" tumours we are describing these cells are much more plentiful and better formed than in the other tumours we have mentioned. They are surrounded by intermediate cells, generally of the usual spindle-form type. Their large proportion of giant cells, together with the peculiar colour of the growths, marks them as a distinct variety. Although these giant-cell tumours generally arise within the bone, yet in one of their favourite seats under the gums on the jaws they show a more superficial connection with the bone, being often united with it by dense bone; in these examples it is difficult to say whether the origin is within or outside the bone. The red tumours are generally not malignant, but they may have in them other ingredients of a malignant nature. Thus, in one specimen a large tumour growing from the fibula is composed of a soft matter and bone; the former consists of a structure which can be called by no other name than medullary sarcoma, while in parts there are nodules of the red myeloid matter. The lad from whom it came died some months after amputation, with the same disease in the lungs and spine, which was also composed of the same three elements.

All endosteal sarcomas are not of this "myeloid" form, however. The latter, as we have said, grow in the end of the bone chiefly. Those that grow in the middle of the shaft of a long bone are generally harder and have less tendency to distend the bone; they rather erode it and thus weaken it, so that it may break and the growth sprout from the fracture, as happens more often in some cases of carcinoma. Similar firm fibro-sarcomata occasionally grow in the body of the lower or upper jaw.

Many very remarkable tumours have their seat in the jaws; besides the carcinomas and sarcomas having their origin in the antrum, we meet with *cystic forms of disease.* Besides the mere expansion of the antrum, simple and dentigerous cysts have been met with. Mr. Eve described a compound cystic growth, which he believed commenced in the gum. These cysts may contain a serous or glairy fluid. When dressing for Mr. Key in the year 1846, we saw him remove the half of the lower jaw from a girl, aged eighteen, in whom the jaw had been enlarging for twelve years. The whole ramus when removed was found to consist of a bony case containing four or five smaller cysts, which looked like expanded alveoli. These contained fangs of teeth. The cells contained a glairy fluid. Fresh bone soon formed in the place of the ramus, and at the end of four years there was very little disfigurement.

Occasionally, as in an instance upon the tibia, melano-sarcoma is found affecting the bones secondary to such melanosis elsewhere.

*Myxoma.*—The gelatinous forms of sarcoma, such as contain mucin in the tissue, are not infrequent in the bones. They often present the characteristic structure of "mucous tissue" or myxoma, but frequently they pass into the consistence of soft cartilage (myxo-chondroma), or they graduate into the consistence of firm sarcomas. When well characterised, they form soft tumours of almost gelatinous consistence and glutinous feel on section. Their substance may be nearly pure, or permeated throughout by threads or networks of bone running through them. They are generally endosteal in their origin, and are affiliated by Virchow to the marrow, which approaches in its developmental stages very closely to mucous tissue.

*Angioma, aneurysmal tumours of bone.*—Much interest attaches to the vascular tumours of bone. A great deal of obscurity still beclouds the subject, though a large number of cases are recorded, and they have been collected and considered by several authorities in the endeavour to reduce them to some clear and simple pathology. Two views have competed in regard of them—one which regards them as essentially aneurysmal, being primary dilatations of the vessels; the other, which considers them to be solid tumours softened down to blood-cysts, or permeated by wide vessels. In favour of this latter view is the frequent presence of small dilated vessels in the outer wall of the main cyst, and the gradation of these pulsating tumours of bone into such examples of osteo-carcinoma and osteo-sarcoma as are highly pulsatile. In favour of the former view that regards them as aneurysms is the fact that some have been cured by ligature of the supplying vessel. The vascular tumours are very rare, and it is not easy to examine them minutely. They are generally nævoid, being composed of dilated intercommunicating channels, separated by fibrous and osteo-fibrous plates and beams, which form their boundaries. The lining of the blood channels being very thin, and like that of the veins in structure, sometimes the channels are large, sometimes they are smaller. Often the search for the materials of actively-growing tumours has not succeeded in discovering in some of them anything which could be regarded as a matrix whose blood-vessels were dilated. They sometimes have clinically a very deceptive resemblance to aneurysm. Thus, when such a tumour arises in the inner end of the clavicle, it may be a most difficult matter to distinguish the case from one of subclavian aneurysm thrusting forward and expanding the bones. The differences that have been noticed in examples of pulsating tumours of bone which we have carefully examined have led us to believe them to be of different natures. Some appear to be truly dilated vessels which are in direct connection with the arterial system, and hence receive the arterial impulse and are pulsatile. Others have a more obscure origin, and appear to be formed by cystic changes in vascular sarcomatous tumours arising in the medulla of the bone; such are those which are formed especially about the knee-joint, where the head of the tibia for instance may be found greatly dilated by a tumour of blood which, when carefully searched, reveals nothing more than blood and blood-clot. It is not, however, only in bones that this difficulty in explanation of blood-cysts is met with; we shall have to draw attention to like instances of obscure blood-cysts in the soft parts, especially the deep parts of the limbs where such blood-clot-like tumours often have a very grave significance from their tendency to prove malignant.

A very remarkable case was reported by Mr. H. Morris of long-standing pulsating tumour on the cranium. There were also others on the clavicle and femur. At the same time the thyroid was much enlarged. The case may be compared with one or two others related from abroad, where there was a growth in the thyroid gland, and secondary cystic tumours of a like character in the bones and other parts.

*Carcinoma.*—The rapidly growing forms of sarcoma often prove very malignant by returning after removal and by metastasis to other parts. These characters of malignancy are of so much greater importance than any mere histological distinctions among tumours that they have always received the chief attention, and have hitherto in practice almost entirely overpowered the attempt to distinguish accurately the carcinomas, *i. e.* tumours composed of an alveolar framework containing epithelioid cells, from the sarcomas whose composition is a homogeneous mass of cells connected by intermediate "intercellular" tissue more or less fibrillar.

Indeed, authorities are just now perhaps farther off than ever from a clear understanding as to the mutual limits of these two classes of tumours in bone, especially since Billroth has proposed to regard some of the tumours of alveolar structure as alveolar sarcoma rather than as carcinoma, on account of the want of resemblance of their cells to epithelium. We believe that this is a true view as to many cases. Thus, in a great tumour of the base of the skull which we recently examined, and which presented on section a minutely reticular structure to the naked eye, we found a very well-developed alveolar framework, and yet the areolæ were filled with cells which were round, and were separated from each other by an intercellular substance. This appearance of areolæ enclosing round sarcoma-cells is often caused by the section having cut bundles of spindle-cells which were running in different directions, some being divided across, while their neighbours around were cut lengthways.

A long time and a great number of observations will be required to settle this question; in the meanwhile it is certain that tumours of a true carcinomatous structure are found in the bones. The most certain examples are those in which several secondary cancerous tumours are found in the skeleton in cases of carcinoma of the breast, or, more rarely, of the uterus. The nature of the primary cancer determines the nature of the secondary. This is a point of great interest in the history of cancer, and one we should bear in mind as of practical importance, that, various as the kinds of malignant tumour are, yet the several growths found in any particular case of multiple malignant tumour are all of the same kind. The tumours secondary to scirrhous carcinoma of the breast are found to be themselves also carcinomatous. They may differ a little in their consistence from the primary tumours, the secondary growths being rather softer and more rapid; but the structure has the same histological character.

Besides such secondary carcinomas of the skeleton we find some that are primary. Thus, we recently examined several cases of true carcinoma arising in the bones of the skull. In one of these the basilar bone was the chief seat of the disease, which had extended to the jugular foramen, and destroyed the pneumogastric and spinal accessory nerves; it was very interesting to observe that in this case the trapezius and sterno-mastoid muscles had wasted away almost completely, notwithstanding that they possess an independent spinal nerve-supply. But most of the cancerous tumours of the skull occur in its vault, in the form of more or less and, perhaps, remarkably numerous patches of growth that often appear to arise in the diploë, and to grow at the expense of the bone until they thrust themselves through one side (generally the inner), or both sides, so as to push in the dura mater, and lift the scalp. When the calvaria is torn off in the usual process of opening the skull, such tumours are often left attached to the dura mater, and so appear to belong to it rather than to the bone; hence it is often not easy to say which tissue they really arose in, especially as they generally begin upon the inner face of the calvaria, and lie at first between the bone and dura mater. However, their origin from the bone is generally shown by their being covered by a complete layer of the dura mater, while the bone is hollowed out by their growth. In the dry bone such hollowed spots may appear as clearly punched holes, or they may be filled up with a network of bone belonging to the growth; the edges are abrupt and show either no diseased change or else a growth of bony papillæ around the hole. These growths of papillæ often form quite a striking feature in the disease, and it is interesting to notice that they are in no degree cancerous, but are the result of the increased blood supply which the cancer has induced in its own neighbourhood. In these cases of cancer of the cranium we will rather frequently find that the spinal column has similar patches of cancer, and, in some cases, the tumours are scattered through the pelvic bones, ribs, sternum, &c.

One example of such disseminated cancer of the cranium and other parts of the skeleton proved to be of an exquisite *colloid* structure. We have also found medullary sarcoma scattered in the skeleton with the same distribution, so that such a dissemination is by no means peculiar to carcinoma.

Carcinoma is comparatively little liable to undergo ossification; nevertheless, we sometimes find the larger trabeculæ of the alveolar framework of the tumours partly changed to bone—osteo-carcinoma.

Besides the origin of tumours within the bones primarily or secondarily in the cases just described, the bones are also liable to be reached and attacked in the spread of carcinoma, and in this way we meet with epithelial cancer running in the bones. Thus, in an example from a patient of Mr. Cock the upper jaw is largely implicated in an epithelial cancer of the cheek, and it is far from infrequent to find a cancerous sore on the leg reaching the tibia, invading it, and extending in its structure. The epithelioid cells of the cancer, as they grow, insinuate themselves into the Haversian spaces, and the bone slowly disappears before their growth, so that in a section taken from the edge of a cancer where growing into a bone we see the remains of the bone reduced to mere spicules, which remain embedded in the new epithelial cells of the cancer; these spicules we may see reduced to the minutest fragments, and it is difficult to find out whether the proper cells of the bone do or do not give rise to the cancer cells. The spicules of bone left embedded in growing cancer commonly show those excavations of their edges which are called Howship's lacunæ. These are found packed full of cancer-cells; but whether by multiplication of the bone-cells we could not assure ourselves. The examination of the growing edge of a tumour in bone, to ascertain the behaviour of its cells, is very difficult. Sometimes the border of the growth shows a great quantity of very small cells, such as are called by Virchow granulation tissue, and these are described as arising by multiplication of the bone-cells. Analogy with the behaviour of the cells of fibrous tissue under similar circumstances would support this view strongly; but we have not been able to convince ourselves of its actual occurrence.

Eve has described a primary *tubular epithelioma* of the upper jaw. It was composed of fibrous stroma, in which masses of cells were arranged in round spaces and grouped together as in tubular glands; the cells were small as in adenoid tissue.

For *osteoid cancer* see *osteoid chondroma.*

*Osteolytic cancer.*—Some examples of cancer in bone are very far from appearing as tumours; they rather give the appearance of local osteomalacia. The interior of the bone at the affected spots contains soft reddish matter in the form of a rounded patch, and there may be openings through the superficial hard layer of the bone leading into this patch. The red substance looks like injected marrow. It is composed of cells which are small and irregular in form and contain one or more small nuclei. We have seen this form of disease distributed extensively in the skeleton, especially in the calvaria. Such cases have been brought before the Pathological Society, and are found scattered through the journals. A very characteristic case is given by Leubuscher. These formations appear to be of the same nature as those called osteolysis by Lobstein; such soft cancer of bones is very apt to be widely distributed, and then is found in the form of rounded patches in the vault of the skull, in the pelvis, sternum, vertebræ, and long bones, having indeed just the same distribution as we have already described as belonging to multiple carcinoma and colloid.

When attacking the bodies of the vertebræ it should be noticed that cancer has the very peculiar effect of shortening the spine, often remarkably, so that the stature of the person is reduced considerably. The new growth occurs in the substance of the vertebræ, and, being soft, is squeezed out by the weight it has to bear, so that several of the intervertebral cartilages meet together, the cancer being forced out to occupy a position around them. Sometimes cancer grows in the laminæ and reaches the nerves, giving rise to a very painful form of paraplegia. Such diffused malignant disease of the bones may cause slow, painful, wasting cachexia. Its presence should be considered among the probabilities in obscure cases with such symptoms.

*Cystoid disease of the bones.*—A few cases are on record, as by Froriep and by Engel, of cystoid change distributed through the skeleton, the bones wasting into cystic cavities so that numerous fractures occur. The cysts begin as red spots, the cystic change follows. When small they have no wall, afterwards they grow greater and become multilocular, lined with connective-tissue membrane, and full of a dark fluid or a pappy material. There is some obscurity about the nature of these cases. They would appear to be allied to those cases of osteolytic cancer to which we have just alluded. But they still more resemble osteomalacia, to which both in clinical features and pathological products osteolytic cancer bears so deceptive a resemblance.

*Cephalhœmatoma* is the name given to those blood tumours which occasionally arise on the heads of children at time of birth. We do not allude to the scalp tumours, which are more common, but to effusions of blood which take place beneath the periosteum, and which is a rarer affection. The blood may be absorbed, but sometimes it breaks out externally, leading to a slough; or it may involve the bone and penetrate to the brain, leading to death.

This last affection leads us to remark that in *scurvy* the blood is poured out, often in large quantities, beneath the periosteum, and that this subsequently hardens, and even becomes vascular; thus Busk has succeeded in injecting such a mass from the vessels of the bone. In a case already mentioned the child was markedly rickety, and therefore it is a question how far these blood effusions are connected with scurvy or rickets.

*Hydatid* in bone is very rare, but it is occasionally met with, as in a preparation of a tibia, in the head of which is an encysted hydatid containing many daughter hydatids. This is a unilocular hydatid, the whole being contained within one parent cyst. We have also two preparations of hydatid of the spinal column. Both of them are instances of multilocular hydatid. There are very numerous hydatids, many very small, extending widely in all the tissues about the spine under the pleura, and also perforating the spine and destroying the vertebræ, raising then the dura mater as a tumour which compressed the spinal cord and caused paraplegia which was fatal through sloughing of the bladder. In these multilocular hydatids there is no common enclosing parent, but the cysts are found budding one from the exterior of another, producing often short chains of three linked together by the yet undivided bud-stalk. Microscopically the tissue of the hydatid, &c., has the characteristic concentric lamination, but there are no hooklets; in other words, the sacs are acephalous. Although hydatid of the spine is a rare disease, yet as many as ten cases have been collected.

There is a parasitic disease affecting the jaw of oxen called *actinomycosis*, to which we shall again refer under "Liver." A somewhat analogous disease called "mycetoma" has been described by Vandyke Carter as attacking the bones of the feet of the natives of India. The feet become enlarged, and covered with sores and sinuses passing to carious bone.

*Tubercle* in bone is still, as always, a theme of contention among pathologists. Some consider as tubercle all the numerous cases in which we find a caseous slowly progressing suppuration tending to the softening down of the proper tissue within the bone. This we have already described under the title of caries. There is much good reason to consider that such caries is of the same nature as local tuberculous disease. In either case the proper tissue is destroyed by a lymphoid formation, which perishes, itself and the proper texture dying together. In either case the disease is slow and tenaciously obstinate. Any one of the whole class of tuberculous diseases, including caries itself, is very often and very significantly associated with others of that class, so that by their concurrence as well as by their analogies and identity of character these diseases form one great class. Thus the scrofulous or tubercular caries of the joints in particular is very frequently associated with pulmonary phthisis and other tuberculous diseases, as of the intestine, larynx, &c. There is then, we think, sufficient ground to class the caseous disease of the bones with the caseous disease of the glands, spleen, suprarenal capsules, kidney, testis, lungs, &c., and if this be agreed, it is comparatively unimportant by what name we may call the group which has common features of such main importance, especially when there are no distinctions among them to be in any way compared with these.

Yet there are a number of the younger pathologists who maintain that true tubercle lies at the bottom and is the starting-point of numerous diseases of the long bones, of the spine, and of the joints. There seems to be an increasing tendency to favour this view. Klein and Lingard have inoculated guinea-pigs with tubercle and have found, two weeks afterwards, the red marrow at the end of the tibia and femur containing small yellow spots. These were true tubercle and were full of bacilli. (See further under "Joints.")

# DISEASES OF THE JOINTS

We shall only briefly run through this section, as the whole subject is given in the surgical lectures. The joints are composed of the articular extremities of the bone, which are cancellous, and covered by a more compact layer of osseous tissue; this, again, has over it the articular cartilage, and the two ends are connected by a capsular ligament, lined by a synovial membrane. Opinions vary as to whether this membrane terminates at the end of the cartilage or is carried over it; but we believe, in the adult, it ceases at the cartilage, the cells which are seen on the latter being its own superficial layer, and not those of a distinct membrane. The synovial membrane consists of a delicate vascular tissue, covered with epithelium, and from the surface of which there hang down a number of vascular fringes, which become enlarged under certain morbid conditions. The diseases of the joints are nearly all either inflammatory, or degenerative, or a combination of these.

**Inflammation.**—*Simple and traumatic arthritis.*—For the sake of classification, the distinctions into the various forms of inflammation, and into acute and chronic, are made; but in particular cases it is often difficult thus to distinguish. The changes which go on in the synovial membrane are very similar to those witnessed in other serous surfaces—an acute process accompanied by effusion of lymph or pus; or a more chronic process accompanied by a simple serous effusion, or by the production of more solid material. *Simple synovitis* is seen best after an injury where the ordinary symptoms and signs of inflammation exist, with swelling and effusion into the joint, and which may subside again in a few days. If the joint can be inspected at this time, there will be found great vascularity of the membrane, with increased serous effusion, and, probably, some flakes of lymph; the latter will be seen covering the synovial membrane, but not the cartilage, except in young subjects, where we have been able to detect a distinct layer of lymph, the part beneath being highly vascular. When the injury is severe, suppuration occurs, the joint becoming distended with pus. This result, which is always very dangerous, is, unfortunately, too often excited by what appears to be a disproportionately small degree of damage, as when a violent and perhaps fatal suppuration of the knee-joint arises from a careful attempt to remove a loose cartilage, or a fragment of bone from it; at other times the severity of the arthritis is only in accordance with the character of its cause, as when a neighbouring abscess breaks into the joint. In any such circumstance the joint is found deep red or purple within, and containing a quantity of pus, generally of glairy consistence from some admixture with synovia.

Should a case of traumatic arthritis be protracted for several weeks before it comes under examination, the cartilage will be found attacked, and at last the bone will become affected. Of course, in some cases the bone is implicated from the first, as in compound fractures into the joint. The mode in which the cartilage is removed differs, we think, in the appearances it presents from the process of its removal in scrofulous joint disease. At least, we have nearly always found in grave traumatic inflammation of the joints sound-looking cartilage standing in greater or smaller patches like islets with abruptly defined margins. These islets are firmly rooted on the bone beneath, and appear to waste away at their edges only; while in tuberculous inflammation the cartilage is found to soften down, and be removed in thin flakes, coming soonest from the surfaces of the bone at the points where they are naturally in close contact.

Most arthritis that is not due to mechanical injury or other obvious cause is reckoned rheumatic, but generally many joints are affected in rheumatism. Trousseau, however, speaks of rheumatic inflammation limited to a single joint, and we sometimes see monarthritis accompanied unmistakably by the conditions of rheumatism; but we equally undoubtedly do meet with simple arthritis of a rather acute and severe character where there is no sign of any rheumatic state about the patient.

*Simple effusion.*—As in the pleura we may meet with a chronic effusion of serum without any attendant symptoms, dependent on a morbid (though unappreciable) state of the serous membrane, so a similar process may occur in a joint, whereby a large secretion of fluid and swelling of the joint take place, producing the affection known as *hydrops articuli.*

*Pulpy degeneration.*—A still more chronic process, of a different character, whereby a solid material is formed, and the membrane altered in character, constitutes the pulpy degeneration of Brodie. In speaking of the periosteum, we said that when an exudation occurs from a membrane and into it, the membrane itself becomes altered and incorporated with the new material; and here, too, by a slow production of lymph from the synovial sac, it becomes swollen, soft, and so changed in form, that from being a thin tissue it becomes a soft and thick one, and then, as the process still continues, it grows thicker and thicker, until this pulpy membrane is produced. We use the word "grows" advisedly, for when the synovial membrane has become vascular, and it is seen to be gradually increasing in thickness, the process is more allied to a growth than to organisation of previously effused lymph. The joint becomes much enlarged and soft, and the disease can be easily detected by the *tactus eruditus* of the surgeon. Although the affection in its simplicity may be looked upon as a special form of disease, we believe lesser conditions may be constantly met with in various morbid states of the joint. Thus in severe cases of general joint disease involving the cartilage the synovial membrane may be found converted into a similar pulpy gelatinous state. Indeed, in our experience it is rare to find the pulpy condition we have described, unless characters of a scrofulous arthritis are present in the joint.

Another chronic diseased condition of the synovial membrane is where a number of vascular fringes hang down into the joint; these appear to be enlargements of the natural fringes described by Rainey. If we look at these various specimens of disease of the knee-joint, we observe this condition, and so also in some others to which we have already alluded.

**Rheumatic Inflammation.**—It is fortunately not often that patients die of acute rheumatism, so that anatomical examination of the joints in that disease is as rare as clinical observation of them is frequent. When an opportunity is offered we are sometimes struck with the slightness of the change within the joint; it is scarcely noticeable; indeed, it would probably not be recognised unless a minute examination were made; the only appearance observed being a slight increase of fluid, the hyperæmic condition of the synovial membrane, which, no doubt, had existed during life, having now disappeared. If we remember how fleeting is the pain and swelling of the joints, and how a part affected acutely at one moment may in a few hours be quite well, we shall perceive a reason why no structural change should be manifested.

But all cases are not of this slight degree; sometimes we find the joint much distended with turbid fluid, and its surface pink from congestion of its vessels. In other cases large flakes of lymph float in the fluid, or a coating of lymph is found lining the whole of the synovial surface of the joint. In some of our specimens we see large masses of lymph which were removed from the knee-joint in cases of acute rheumatism. Again, it is not only within the joints that we see evidence of inflammatory action in rheumatism, but also in the *sheaths of the tendons*; we have several times found the sheaths of the extensors, where passing the wrists, full of the same turbid flaky fluid as was present in the wrist-joints.

This considerable alteration in acute rheumatism enables us to understand how it is that recovery is often so slow and incomplete. It is true that the behaviour of the joints after acute rheumatism is usually in contrast with the behaviour of the heart when it has been affected—a contrast that is sad enough on the part of the heart, which so frequently remains permanently affected, while the joints entirely recover their suppleness and freedom (we shall see the reason of this when treating of rheumatic heart disease), but, although rarely, rheumatic joint disease does leave severe after effects. The ligaments contract so much that the joint is permanently stiff; or the synovial membrane is so damaged by inflammation that it will not reabsorb the fluid effusion, which consequently remains as a long-standing hydrarthrosis; or even, in some cases, an adhesion forms between the articular surfaces by bands of fibrous tissue.

There is another set of conditions connected with rheumatism in which a yet more intense joint disease may be sometimes observed. Thus the cardiac inflammation of a rheumatism may become ulcerative, and at the same time the joint disease may put on a destructive severity, the cartilage being removed, and a state of simple caries or ulceration of the bone established.

The post-mortem examination of simple chronic rheumatism is meagre in its results; such cases are found in women especially, more or less distorting the fingers, and often called by the name "rheumatic gout," which usually covers a doubt its employer cannot solve. The amount of distortion would lead us to expect considerable changes within the joint, whereas we do not generally find the bony surface at all altered; the synovial membrane looks healthy. The only discoverable change as cause of the distortion is thickening and contraction of the ligaments, but we know it is not easy to see where ligaments are a little thick and contracted. The swelling we see around such joints in the wards is the results of œdema from one of those frequently recurring inflammations which make these cases so intractable.

We might here make mention of the small nodules found in the skin in rheumatic subjects, and first described by Dr Barlow under the name of "subcutaneous fibrous nodules." They consist of a simple network of nucleated fibrous tissue.

**Pyæmic and Gonorrhœal Inflammation, &c.**—The most severe forms of inflammation of the joints are seen in connection with pyæmia. In this disease a dirty thin pus may be found in the joint after it has been affected for only two or three days; but as yet there is no destruction of tissue, the synovial membrane being only found somewhat more vascular; if, however, the patient live, a destruction of the joint would very rapidly ensue, the synovial membrane being thickened, and the ends of the bones denuded of cartilage. This we have often the opportunity of seeing by comparison of the joints that have been surgically opened, with those that have been left to their own course, through the surgeon's despair of relieving by further interference. Thus, in a case we had not long ago, the right shoulder was attacked after the left. The left, which was first attacked, had been opened, but the right, which swelled when the patient's case was thought to be obviously beyond relief, was left unopened. The contrast of the states of these joints was very instructive. The left shoulder-joint, which had been longest diseased, but had been incised in time, showed no disorder beyond the confines of the joint itself. But the right showed a most frightful destruction, indeed, the scapula lay bare in a bath of foul pus. Such instances, not always so striking, we occasionally see, proving the importance of early giving free exit to the pus from within the joint.

Attention must be directed to the relations which the polyarthritis of rheumatism has to the polyarthritis of pyæmia. In both diseases the heart is apt to be affected. There is reason to think that the polyarthritic variety of pyæmia is a disease that differs from that variety which especially affects the lungs, liver, &c., and is a disease more amenable to treatment. In speaking of acute necrosis, we pointed out a curious variety of pyæmia which too often accompanies it, affecting the heart and kidneys in its secondary suppurations almost always, while the joints generally escape.

In speaking of *gonorrhœal* rheumatism, we should first note that in two cases which have occurred at Guy's an obscure fatal pyæmia proved to have for its cause a gonorrhœal inflammation of the urethra, the evidence of this being the discovery of purulent inflammation of the prostatic plexus of veins, while the urethra was full of pus. *Gonorrhœal* rheumatism is usually a subacute form of a pyæmic inflammation. We mention it here in connection with pyæmia, not because pus is formed, for this we think is very rare, but because we think it owns a similar cause. It is only fair to state that opinions vary much as to the existence of this form of disease; but our own experience leads us to the conclusion of its frequency, and this is supported by the fact, that we believe similar affections of the joints may be witnessed in women and other persons who are suffering from purulent discharges. Gonorrhœal rheumatism is characterised by a subacute inflammation, affecting sometimes the knee and larger joints, but more especially the wrist and ankle, more commonly the latter; and when we speak of this, we mean not only the true ankle-joints, but all the smaller tarsal joints in the neighbourhood; these deep-seated joints, with the ligaments, become affected, and a very chronic painful disease is the result, productive of a thickening and induration of all the articular tissues.

Mr. Lucas has described an acute synovitis of infants who were the subjects of purulent ophthalmia. He regarded the affection as of a pyæmic character.

Mr. Tom Smith has also described an acute arthritis of infants going on to the destruction of the joint; suppuration occurring with ulceration of the bone and cartilage. In early cases he has found the synovial membrane thickened, and covered with lymph, and absorption of the cartilage commencing.

Mr. Morrant Baker has also observed a destructive inflammation of the joints in children, beginning apparently as an acute epiphysitis.

Some surgeons have spoken of synovitis as occurring in syphilis, and we certainly have observed a temporary effusion in the joints of those who are being treated for this disease.

Along with gonorrhœal rheumatism we should consider *scarlatinal* rheumatism, in many aspects so analogous. Scarlatinal rheumatism is often nothing less than severe pyæmia, and evidently traceable to purulent infection from the sores in the throat. But all scarlatinal rheumatism is not of this grave kind; we not infrequently meet with little children recovering from moderately severe attacks of scarlatina, during which no loss of tissue had occurred in the throat, but they complain of their joints, which become red, and swell, showing that a distinct synovitis has taken place; afterwards the swelling goes down, and no evil comes of it. Also it will be remembered here that these rheumatic sequelæ of scarlatina sometimes induce, among the other local disturbances, acute endocarditis.

*Puerperal* rheumatism stands in a very similar relation to pyæmia, but suppuration is relatively more frequent. The same is true of that following *smallpox.* Besides these, we occasionally meet with a class of obscure cases, either in the wards or on the post-mortem table, in which polyarthritis of more or less grave kind is associated with pellicular inflammation of the fauces, and it is difficult to say whether the disease is to be regarded as diphtheria with rheumatism related to it like it is to scarlatina, or as rheumatism with pellicular pharyngitis. In one such case, two or three years ago, in the Clinical ward, the tongue became much swollen, and on one side of it there formed a considerable slough; yet, after much danger of suffocation and severe articular inflammation, which did not reach suppuration, the patient, a stout, flabby, middle-aged man, made good his recovery.

In short, the connection of polyarthritis with febrile diseases is a very interesting subject, and would well repay more special inquiry than it has yet received. It may be generally said that any of the fevers may be accompanied, or more generally followed, by rheumatoid polyarthritis. The occurrence is rare in the case of some of the fevers. But with others it is so frequent that it becomes important to be aware of it. Thus, some epidemics of *dysentery* have been marked by the frequent occurrence of rheumatism as a sequela of the intestinal disorder in the subjects of the attack. This connection of dysentery with rheumatism was observed very long ago, and more recently attention has been drawn to it by the Norwegian epidemic a few years ago, reported by Homan and Harting, and by some still more recent instances. In some of these cases endocarditis accompanied the rheumatic disorder.

**Scrofulous Inflammation, or White Swelling.**—The remarks we have already made in reference to tubercle in bone will prepare us for the difficulty of knowing what is exactly understood by scrofulous affection of joints. The old opinion was that tubercle formed in the bone, and then softened, and set up mischief in the neighbouring joint. This opinion, which was for a long time discarded, is now once more coming into favour. This is owing to the fact that the material once called tubercle, having been regarded as inflammatory lymph, is now again receiving its original name owing to its histological characters, and from the presence of bacilli within it. Until the revival of the tubercular theory it had been taught that white swelling was a synovitis of a slow nature occurring in scrofulous children as in adults, and the articular ends of the bones became involved in the inflammation. It is not often that we meet with a chance of examining scrofulous disease of the joints in its earlier stages, but in a case very lately a knee-joint of an amputated limb, which had been removed for caries of the head of the tibia, showed in its synovial membrane tuberculous ulceration in a most interesting way, lenticular ulcers, of a size from millet seed to hemp seed, being distributed closely and regularly over the synovial membranes, which otherwise was little altered. The joint was only secondarily implicated in the disease. This, however, although it suggests, yet would not prove such tubercles to be present when the knee-joint is the primary seat of the disease, for we shall often have to mention the frequent formation of tubercles close about old caseous disease, when there is no opportunity of proving this caseous disease to be itself tuberculous; and we shall find that several conflicting interpretations are put upon this association, which interpretations we shall allude to on a more convenient occasion. Similar cases of tubercle in the synovial membrane have been recorded by other observers.

In the year 1881 Mr. Croft brought the subject before the Pathological Society. His specimens showed the usual granulations growing from the cartilage and elsewhere, but besides this he pointed out a distinct layer of a new formation which he styled tubercular. It was composed of a loose, delicate reticulum containing small cells, together with some giant cells. This, the author maintained, corresponded to tubercle in every respect as seen in other organs; in two of his cases the bone also contained a similar growth. Out of the six cases which he brought forward he was able to follow the course of four, and these subsequently died of tuberculous disease. When first seen they were the subjects of joint diseases only, and therefore he believed that the tubercle was primary in the joint, and was the source of the infection.

The opinion that these affections of the joints are tubercular is supported by Barker and Macnamara, although these two surgeons would place the focus of the disease in the bone rather than in the synovial membrane. They believe that tubercle first forms in the head of the bone, that softening then takes place beneath the cartilage, that the latter then ulcerates, and the whole joint is involved. Macnamara goes so far as to say that 80 per cent. of cases of hip-joint disease begin in the head and neck of the femur, or immediately under the articular cartilage. A primary affection of the synovial membrane or acetabulum he believes to be rare; when occurring it has its origin in injury or pyæmia. There have been described, however, by good surgeons, cases of the worst form of joint disease called white swelling, where, although the cartilage was found diseased, it was thought that the primary affection was in the synovial membrane, this being thickened and covered with flakes of lymph, and its fringes studded with tubercles.

In reasoning from the rarity of the discovery of tubercles, we must remember how unusual it is for the early stage of joint disease known to be scrofulous to come under anatomical examination. But the later stages of the disease are amongst the most frequent objects of our morbid anatomy. In these, if extreme, we find the soft parts of the joint excessively diseased; the interior of the joint being brought to a state resembling that of the sinuses which communicate between its cavity and the outside of the limb, that is, like the interior of very old abscesses. The synovial membrane, or rather what was the synovial membrane, shows degrees of that pulpy degeneration which we have before described, but it is deep purple or red on its surface for the most part, and may be in many parts eroded. The tissue outside the joint is more or less consolidated into a white fibrous mass, soaked with serum, and showing the remains of fat and muscle, &c., when looked at closely. The tissue is in places charged with inflammatory products which may break down, forming abscesses that burst into the joint, or burst upon the surface of the limb, so causing sinuses unconnected with the joint. The cartilage is in such old cases nearly all destroyed; the destruction shows itself, first, where the bones naturally press against each other in the actions of the joint; the cartilage generally is gradually thinned away there, and when reduced much may be found as a thin pulpy layer, separating as a flake from the surface of the bone.

But sometimes, when the joint is examined at an earlier stage, the cartilage is found parting from the bone, as though it were shed like cuticle, or in slower cases the medulla of the bone sends up vascular granulations that pierce the cartilage in many parts, so that it looks porous, meantime softening down while the granulations project in the joint, and meeting with similar granulations may unite and ossify, producing ankylosis. In these cases the bone is observed charged with red lymph, and then the inflammation of the bone is to be regarded as the main disease which implicates the joint through contiguity. This, as we have said, has been thought by some surgeons to be the case universally. Tuberculous inflammation of the bone softening down was supposed by them to be always the starting-point of a mischief that afterwards implicated the joint, and there are those now who believe this to be the case in the majority of instances. For those examples, wherein the disease has not gone on to the utter demolition of the components of the joint, and where a considerable amount of cartilage is left still suffering the destructive processes, show good evidence of the cartilage being partly taken away off the free surface. Thus, we have often seen examples of the fact noticed by

Sir Everard Home, where the cartilage on the borders of the faces of the joint are grooved by the fringes of swollen synovial membrane, which project between the opposed cartilaginous faces; without giving unqualified support to the opinion that these synovial fringes absorb away the cartilage, it is clear that we must consider the removal of it to be so far effected by a process belonging to the inside of the joint, and not to the bone beneath. Probably the pressure of the fringes, swollen and inflamed as they are, causes the disintegration of the parts subjected to it, while the inflamed state of the cartilage itself favours the change. This is rendered very likely through a fact we have already mentioned, namely, that the cartilage goes most extensively and decisively off the parts of the joint's surface which are most firmly pressed together, such as the convexities of the condyles of the femur, and the opposed parts of the tibia, while it lasts very long on the facets of the patella and the other portions of surface which are little compressed; we hence see how pressure will take the cartilage away. As further proving that the disease often begins within the joint, we find the bone around, in many cases, to be simply softened, pale, and very green in texture. Its state, in short, is exactly that we have described as found in the muscles, &c., outside the joint, a state, that is, of wasting away from disuse, so that it is charged with the watery, fat, and œdematous fluids that generally surround the stagnant neighbourhood of old weak inflammations. But such cases as affect the bone chiefly and primarily show caries, and perhaps necrosis, of its articular ends, which are eroded and generally dense, with evidences of active repair showing themselves in a quantity of spicules of new bone, that project in sharp points around the ends of the bones, following the course of the ligaments which they generally are formed in; or the articular faces will be covered more or less completely with new bony matter, tending to repair the mischief by ankylosis.

**Chronic Rheumatic Arthritis** is a name which the authority of the most careful and laborious describer of the disease (Dr. Adams, of Dublin) has rendered classical. This name sufficiently well denotes the main characters of the disorder. It had been called *nodosity of the joints* by Haygarth, which was not an unsuitable name. When affecting particular joints it had been known by names whose application is either partial or not distinctive, such as *malum coxæ senile*, which would wrongly infer that the disease does not attack young people, or *arthrite seche*, which would not concur well with the fact that at some periods of the disease there is often great effusion of fluid into the joint, or *arthritis deformans*, but deformity of the joints is produced at least by gout also. Cruveilhier, who appears to have been the first to claim for this disease a clinical recognition, proposed to call it *usure des cartilages articulaires*. But this name puts forward the erosion of cartilage much beyond its true importance in the disorder

Such erosion is common in advancing life, when there is no trace or suspicion of the disease in question. However, as we have said, the name chronic rheumatic or rheumatoid arthritis is now universally accepted for this disorder, when recognised by its proper anatomical characters; though during life we often hear used for it the vague name "rheumatic gout," which serves to cover over its employer's doubt of the pathology of his case. Sir B. Brodie introduced gouty deposit as a feature of its anatomy, but when such deposit is found it must be regarded as a complication.

The disease has more than a full share of the vagueness of definition of rheumatic disorder, for the chronic and obscure cases of rheumatism are those that especially tend to fall into it. Very often there is no reason to consider the case to be rheumatic, as when it arises from a sprain or concussion to a single joint. But in many cases it has followed out of rheumatic fever in such a way as to appear to be a chronic continuation of that disease, just as chronic pleurisy is a continuation of acute pleurisy. Some choose to interpret this so that the rheumatic fever is held to have been not true rheumatic fever, but "acute rheumatoid arthritis." This is, however, entirely arbitrary. As the cause of this disease is obscure, so also its earlier stages are not well defined; it is only in the later development of its effects on joints that it becomes clearly recognisable. The joint is sometimes described as dry in the first stage, but this statement is, we think, made on account of the dry grating feel which the eroded cartilages of elderly people impart to the hand during movements of the joint. This erosion, however, does not constitute rheumatic arthritis. In its earlier stages the joint shows only the condition we have already considered as *hydrops articuli*. The cavity has in it an excess of clear synovial fluid, while the wall of the joint is more or less thick, and its flocculent processes are increased in number and size, and more or less deeply red from congestion. It is thought that by this effusion the ligaments are stretched and lengthened so as to favour the partial dislocations which sometimes occur, but the dislocations follow upon such changes in the ends of the bones as sufficiently explain their displacements.

It is at a later stage that the peculiar characters of the disease show themselves. These affect chiefly the bone and its cartilaginous covering. When the disease is characteristically established, the joint is seen to be much deformed, the most striking peculiarity being the production of a quantity of new bone in nodular masses around the edge of the articulation, and occasionally of separate pieces in the capsular ligament. One may form an idea of the appearance of the joint in this advanced stage by supposing that its cartilage should be removed, and the ends of the bone made so soft that they partly rub down into a bony paste, which is squeezed out from between them to solidify in the form of a rough ring, turned over all around the edges of the worn and polished articular face. If, then, the capsular ligament be conceived to be thick, and the synovial membrane thickened and here and there flocculent and congested, or provided with polypoid, fatty, cartilaginous, or osseous growths, a very good idea of the superficial characters of the disease will be obtained. The neighbouring bursæ are generally implicated, and add to the deformity and disablement.

The behaviour of the articular cartilage in this disease has received the great share of attention, but we think its importance has been exaggerated. Thus, it is said that the disease really consists of an irritative proliferation of the cells of the cartilage, producing a softened and overgrown state of its substance. The softness then, it is said, induces a wasting of the parts subjected to friction and pressure, while the soft overgrowth of cartilage sprouts at the edges of the joint where it is not repressed, and bulges out in the form of an irregular rim, which subsequently ossifies, and so the bony masses around the joint are accounted for. But the worn and eroded state of cartilage found in chronic rheumatic arthritis is very far from being proper to that disease; indeed, erosion of cartilage is very frequent. It may be said that after the thirty-fifth or fortieth year it is usual to find such erosion, though chronic rheumatic arthritis is a comparatively rare disease. It might even be fairly suggested that the constant presence of this erosion of cartilage in rheumatic arthritis is only a coincidence from both disorders belonging to the years of declining life. But the histological changes are the same, whether the erosion is with or without the changes in the other parts, and it would, therefore, seem very reasonable to think that the implication of the other structures of the joint comes about after the destruction of cartilage has progressed beyond a certain limit so as to throw the wear and tear upon the bony tissue unprotected by cartilage, and so arouse in it and its neighbourhood subinflammatory reaction, thus constituting rheumatic arthritis in its more obvious form. Nevertheless, though this view is plausible, we think a large number of observations induces the belief that the erosion of cartilage does not lead so simply on to pronounced arthritis, but rather there is some other factor which is required to set up the inflammation, such as rheumatic fever or a blow or sprain; and this latter factor is able to set up the rheumatic arthritis in the absence of degenerative erosion of cartilage, as is proved by the not very infrequent occurrence of that disease in the earlier years of life.

When the cartilage is removed the exposed bony surfaces polish each other by mere attrition; their faces being by this means compressed and condensed and the earthy matter is then brought to an even shining surface, bearing often a beautiful polish, as though glazed or enamelled. This layer of enamel, when examined microscopically, is confused and almost structureless, so that it evidently is not a new development, but is rather due to mechanical condensation of the old bone. By this rubbing down and condensation the ends of the bones are gradually worn away, so that, as in our specimens of hip-joint, the head of the femur may be seen partially ground off, the ligamentum teres entirely removed, the articular face flattened and surrounded by a fringe of new osteophytes about its edge; also the rim of the acetabulum is partly worn away, so that the cavity is shallow, while it has a rough irregular rim of new bone around widening it; both surfaces are polished brightly where they have worked upon each other, thus supplying one quality of the departed cartilage—its smoothness—while the other—its resilience—is entirely wanting.

In one case, a longitudinal section of the neck of the femur, shows that the flattened mass of bone, which looks like the head of the bone deformed and pushed down on the shortened neck, is really new bone formed around the stump of the neck of the femur, the head being nearly all ground away. Thus, it would appear as if the triturition, which we just now spoke of in illustration, really had occurred, and no doubt the wearing away of the bone is such a simple mechanical process, but we do not think the new bone around the worn head is formed by anything bulging from the joint, as Rindfleisch's description appears to suppose, but rather we regard the new bone as due to the chronic irritation of the periosteum and other fibrous tissues in continuity with the bone, such bone arising from the chronic inflammation ("ossifying periostitis") in the same way as spicules of new bone arise around the ends of bones in caries of the joints. But in chronic rheumatic arthritis, when compared with caries, the new bone differs characteristically. We shall see by comparing specimens that in the rheumatic bone the new formation is in the shape of rounded nodules of dense consistence and close surface, while about the carious joint the new bone is in fine spicules and needles formed of open-textured bone. This difference is due to the inequality in the rate of the processes which produce the new bone, the slower product of rheumatic arthritis having time to undergo that condensation and smoothening of its surface, which ultimately occurs in all bone formations.

In the *shoulder* the disease has characters very similar to those seen in the hip, as we observe in our specimens; the head of the humerus is quite altered, for instead of being round it is converted by new bone into a large irregular mass, having a polished oval surface on one side, where it meets the glenoid cavity; also, if much enlarged, it has another polished surface above, where it meets the under surface of the acromion; and sometimes even a third, to meet the clavicle. The glenoid cavity is expanded and flattened, but generally has not much new bone around it; probably the greater motion of the shoulder, as compared with the hip, prevents this; but in the capsular ligament there is some bony deposit; the under surface of the acromion is polished by the humerus, and this often is detached, so that it then is difficult to say whether it is the epiphysis separated, or a fresh development of bone; thus there is a distinct square portion of bone attached to the end of acromion by a ligamentous structure, and appearing exactly as if the point of this process had been broken off. We wish particularly to draw attention to this, because only the other day a specimen was sent to us as one of fracture of the acromion, which was really nothing more than an example of chronic rheumatic arthritis. Two or three other similar specimens in the Museum were formerly labelled as fractures. The cartilages are always destroyed, and the articular ends of the bones become polished or eburnated, and just as the ligamentum teres in the hip is altogether lost, so the long tendon of the biceps is often destroyed, or degenerates into a fibrous band. It may obtain a new origin in the bicipital groove. As a consequence, the head of the humerus may become dislocated; then it is that examination of the joint, after death, may leave a doubt as to whether the luxation has been the result of disease, or whether an injury in the first place did not set up the arthritis. In many cases of supposed dislocation there can be no doubt that the disease is simply chronic rheumatic arthritis; but, at the same time, this affection may be caused by injury, as some of our specimens show; though it is doubtful whether the dislocation occurred at the first, or was the result of subsequent changes.

In the *elbow* this disease is found less frequently than in the hip and shoulder-joints, but has the same characters. The cartilages are gone, the ends of the bones are polished, a quantity of new bone is seen surrounding the head of the radius and following the edges of the olecranon and coronoid processes, as well as the front and back of the condyles of the humerus. In one specimen these elevated ridges could be felt during life, and much impeded the motion of the joint, suggesting ankylosis, which, however, never or very rarely occurs in this disease. The capsular ligament also has some separate portions of bone in this case.

In other two specimens you will see in the fossa of the olecranon small bones or ossicles; but whether due to this form of disease we cannot say, as there seems no affection of the joint.

In the *knee* also the same changes occur; rims of new bone form along the edges of the condyles and around the head of the tibia. The deformed femur may grind away the head of the tibia on one side chiefly, as in this preparation, or otherwise wear it into curious shapes. The semilunar cartilages disappear, and the ligaments are so weakened that the knee-joint may become capable of many movements besides the two it is organised to go through; all being equally painful. The productive activity of chronic rheumatic arthritis is often more strongly manifested in this joint than in any of the others. It is not uncommon to find, besides the usual osteophytes on the bones, a number of large or small plates of bone in the capsule of the joint feeling like supernumerary patellæ. It is in this joint too that the growth of projecting and pendulous nodules from the synovial surface is most plentiful and free, as we might expect, remembering how large a share of papilliform projections and fringes it has within it in the alar ligament, &c., such hypertrophy of the synovial fringes constituting often the great peculiarity of the disease. Such pendulous bodies may be either fatty or cartilaginous in structure or they may undergo ossification, and they sometimes separate from their original attachments, and are found as loose cartilages in the joints. This is an ordinary origin of such loose bodies, to which we shall presently advert more specially.

The other joints are also liable to suffer from this disease; the hands and fingers are often much distorted and disfigured by the nodulated, stiffened, and partly dislocated state of the *metacarpal* and *phalangeal* joints. In the *vertebræ* there may be seen in some of our specimens a similar form of disease; new bone being produced along the edges of the bodies, which thus overlap each other, while the articular surfaces are expanded and polished. In the *pelvis*, also, may be seen a chronic affection of the joints resulting in a deposition of bone around the edges of the sacro-iliac and pubic articulations.

Chronic rheumatic arthritis generally does not lead to suppuration of the joint. But an exceptional case of disorganized knee-joint was exhibited at the Pathological Society by the late Mr. Bruce, who had given great attention to the disease. In this case the joint had suppurated, although in its other conditions, especially from the quantity of nodular new bone around the articulation, it had the characters of chronic rheumatic arthritis. Sometimes, also, amputation of the thigh has been required to prevent the threatening supervention of suppuration of the knee-joint. Our experience concurs with that of Dr. Adams when affirming that ankylosis is a very rare result of chronic rheumatic arthritis. Sir B. Brodie says ankylosis sometimes occurs, but the disease, as described by him, is not free from mixture with cases of a different kind.

Mr. Arbuthnot Lane has for many years been studying the changes which take place in the bones, and more especially at their articular extremities, due to the wear and tear of life and to the various avocations in which the subjects were employed. Many of the conditions known as chronic rheumatic arthritis in the long bones and in the spine he attributes to wear and pressure. It is in age that he finds well-marked changes in the hips and other joints. For example, in the temporo-maxillary articulation he finds the fibro-cartilage removed, or thinned and perforated; the articular ends of the bone being flattened and roughened. He considers also that many of the curvatures and misplacements in the spine are produced by the carrying of loads, since he has observed that these curves vary according as the subject of them has borne loads on his head or on his back; or on one shoulder rather than on the other.

In some the spine may not be curved, but sink in on one side, arising from wasting of the body of the vertebra and cartilage on that side. From the same cause a fusion of the vertebræ may have slowly taken place. Mr. Lane thinks that in this way may be explained the peculiar malformation studied by Neugebauer under the name of "spondylolisthesis," where the last lumbar vertebra projects forward over the sacrum. He thinks constant pressure and weight of the body sufficient to account for the displacement without having recourse to the idea of inflammation and rheumatism. The outgrowths around and on the sacrum are equally explicable on this theory. He also explains the great thickening of the supraspinous ligaments, with their ossification, and the bridging over with bone of the sacro-iliac articulation. The subject of course requires much more investigation, and it may be that both the older theory of disease and that of Mr. Lane are right; that mechanical causes may determine the part of the skeleton to be affected, while morbid conditions of the body, gouty or rheumatic, may produce the changes which we see.

*Charcot's disease.*—Under this name we refer to a disease of the joints and articular ends of the bones, which has hitherto been regarded as chronic osteo-arthritis, but which Charcot and his followers affirm to be a disease *sui generis.* This distinguished physician believes that the morbid alteration in the bones is due to nerve influence, and although this special form of complaint under consideration occurs in tabes dorsalis, yet that other forms of spine disease may affect the nutrition of the skeleton, and that there is therefore a true "neurotic arthropathy." Charcot has described the peculiarity of the affection, occurring more especially in ataxia, as a destruction of the joints by an atrophy of the articular ends of the bones and of the parts included in the joints. For example, in the knee the condyles would waste and the head of the tibia shrink in size, and at the same time the cartilage would disappear so that the ends of the bones would rub together and perhaps become dislocated; the patella also would become loose from wasting of the ligament. The limb might then be flung about like a flail. In some instances the capsule may be thickened and some new bone formed in it, and so it may come to resemble the chronic osteo-arthritis. It is remarkable how all signs of active inflammation are absent, such as heat and pain. Opinions vary very much with respect to its relation with the last-named disease; whether it be the same or allied to it, or altogether different. Some have thought that both forms of joint disease arise from a general or constitutional cause, but in one case there is an attempt to repair the atrophied joint by the formation of new bone, whilst in Charcot's disease this tendency altogether fails. The great fact to be observed is the wearing away of the articular ends of the bones, as in a case exhibited by Mr. M. Baker, where the end of the tibia had at last assumed the appearance of the external malleolus. Buzzard, who believes in its essential peculiarity, quotes from Charcot "that it is due to an influence of the nervous system, which makes the bones fragile and leads to spontaneous fracture, and that the wearing away of the articular extremities distinguishes it from common arthritis. In no cases are there hyperostoses or epiphysial hypertrophies."

**Gouty Arthritis.**—After a person has had but a few attacks of gout, the joint affected, usually the metatarsal joint of the great toe, shows a quite characteristic result in the presence of urate of soda in the cartilage. This manifests itself as a patch of opaque white, like chalk; when this is small the cartilage may be but slightly affected in its nourishment by it; but if there is much of the chalk present the cartilage will be found thin and worn looking. Sometimes the chalky substance is seen deposited in eroded cartilage, but then we think the erosion and the deposit are really independent though coincident. In severe cases the urate not only charges the cartilage but is found in the ends of the bones, as in a specimen from the great toe. Also it will accumulate among the ligaments and cellular tissue outside, forming the well-known "tophi" or pasty concretions, entangled in the meshes of areolar tissue, which we see in our patients. It appears that when once this urate of soda is deposited in the cartilage it does not afterwards disappear, and thus it gives a very useful means of assuring ourselves of the former occurrence of gout when there is no history of it at the post-mortem examination.

The gouty condition of cartilage is sometimes found in the large joints. One preparation of knee-joint shows the whole surface, condyles, patella, and tibia, covered with white matter like mortar, which was proved by Dr. Rees's analysis to be urate of soda. It would be well to notice here, however, that occasionally phosphatic paste occupies the interior of the joint, and has been mistaken for gouty deposit. Dr. Pye-Smith has observed that, if patients have died with an active gouty inflammation of a joint, the synovial membrane is very much congested, and in some cases hæmorrhage has taken place into it.

When the gouty cartilages are microscopically examined, their cells are found to be surrounded by needles of urate of soda, crowded around the cartilage cells so as to give them the appearance of mossy birds' nests. In the further stages the crystallization extends through the rest of the cartilage, and, as we have said, invades the bone and tissues outside, but it always begins in the cartilage of the joint. The joints appear liable to it very much in direct proportion to the severity of the mechanical wear and changes of temperature they are subjected to. The metacarpo-phalangeal joint of the great toe is by far most frequently affected; this suffers most among the joints of the foot in walking, as we may see by turning to look at the imprint of our naked foot on the sand. The influence of cold is probably instanced in the case of the external ear, in which we may find gouty concretions, so that in deciding on the gouty nature of a case we are often much helped by finding these concretions in the auricle. They frequently arise without any pain.

It is not usual for a gouty joint to show the thickening of the ligaments and development of bone, &c., which characterise chronic rheumatic arthritis; indeed, in their complete development gouty and chronic rheumatic arthritis are very different from each other. But, as we have said, we sometimes meet the urate in cartilage which has undergone erosion, and we must remember that some consider erosion of cartilage to be proper to rheumatic arthritis, although we do not join in that opinion, but rather believe that erosion of cartilage belongs to elderly life just as deforming arteritis does. It certainly must be very rare to find gouty deposit complicating advanced and characteristic rheumatic arthritis, for we have never met with it. The diseases affect, on the whole, different sets of joints, rheumatic arthritis being best characterised in the greater joints and gout in the smaller.

**Dislocation.**—This is fully treated of in the surgical lectures, and therefore we will merely refer to our specimens, showing new sockets in *unreduced* luxations. Many of these show a surprisingly good substitute for the natural joint ; a dense fibrous tissue takes the place of cartilage. The joint is made smooth by a new bursa, and surrounded by dense areolar tissue composing a fairly efficient capsular ligament. Now and then we may meet with the affection known as *congenital* backward dislocation of the hip-joint. The child walks in a peculiar, stiff, upright manner from the difficulty of balancing the trunk on the thighs, and has a rolling gait. On examination it is found that the head of the thigh-bone constantly falls out of its socket. This is owing to a badly formed acetabulum, or to the absence of, or, perhaps, lengthening of, the ligamentum teres. It is possible that it may be due to a disease in fœtal life, as already seen in the sacro-iliac synchondrosis of the oblique pelvis ; but of this there is no proof. In a few cases where an opportunity of examination has existed there has been found either old disease, or the capsule has been very large and the ligamentum teres very long. We sometimes meet with persons who are constantly having a particular joint dislocated ; this probably arises from injury in the first place. In the head of a femur which came from a woman who had had her thigh dislocated twenty-two times the ligamentum teres was quite destroyed. Dislocation occurs from progressing *disease.* Thus, in the hip the head of the bone, or what remains of it, slips out of the socket on to the dorsum of the ilium. In the knee, when all the ligaments are destroyed, the bones are drawn out of their place and, like those of the hip, take a definite direction. The tibia is pulled outwards as though by the popliteus muscle, so that the outer condyle of the femur rests on the middle part of the head or on the inner tuberosity of the tibia, and the patella becomes attached to the front of the outer condyle. We must be careful not to confound a simple dislocation and its supposed results with dislocation the consequence of disease, as has often been done in the case of the hip and shoulder.

*Dislocation of cartilage* sometimes occurs as a partial detachment of a semilunar cartilage in the knee-joint.

**Ankylosis and Synostosis.**—Ankylosis is a term which was formerly used as synonymous with stiff joint, originally implying that the joint was bent at an angle. Now, such unions in the joints are distinguished into two kinds: first, those dependent on a mere ligamentous union between the ends of the bone; and, secondly, those in which the union is effected by bone: the former class are called *ankylosis*, and the latter *synostosis;* what is called *false ankylosis* is mere immobility of a joint dependent on a stiffness of the ligaments and other parts external to it. Synostosis may generally be looked upon as a further stage of ankylosis, following upon a greater destruction of the joint; for if any cartilage be left when the disease ceases, or even if the articular lamella of the end of the bone be quite perfect, the union is merely by fibrous tissue, and in such cases some motion is still left. Where a good bony union is formed the disease has run such a course as we described in speaking of the severer inflammations of the joints, especially traumatic and strumous. The synovial membrane is replaced by a soft gelatinous matter, and at the same time the cartilage disappears, so that an examination of the joint shows the ends of the bones covered with granulations; and as the ends of the bones themselves have, no doubt, been participating in the change, a slight disintegration of the whole surface has taken place. From this the formation of vascular granulations has occurred; and now, if the active morbid process ceases, and the irritation descend to the degree compatible with formation of a new tissue, ossification takes place, and the two surfaces are united. In some cases of synostosis it is probable that the union has not been completed in this simple way, but that the original shafts having disappeared new bone has taken their place, and the part corresponding to the joint is formed from one piece. Thus, one specimen of ankylosis of the elbow does not show any traces of the original bones, but the single massive piece forming the angle seems altogether new. In other cases, however, you can clearly trace the outline of the original bone; in one the head of the radius has remained free, while in others it is ankylosed in front of its natural position. In the knee both ligamentous and bony ankylosis may be met with, as also in the hip; in the latter we may often trace the irregular outline of the original diseased acetabulum fitted to the irregular process formed from the neck of the bone, the head having been lost. In the tarsal and carpal joints we may see in our several preparations that the most perfect bony union may occur. A remarkable disposition to ankylosis is seen in one preparation, showing the bones of the spine, pelvis, and lower limbs all united; there being an evident inclination throughout the body to a destruction of the joints, and a running together of the whole bony skeleton. We have a very rare specimen of a skull of a black man, showing ankylosis of the lower jaw.

A very peculiar form of synostosis is that of the spine, or *spondylitis deformans.* All the articular surfaces of the vertebræ are united, forming the spine into one solid column. The case is quite different from that already referred to, where there is ossification of the ligaments or chronic arthritis, for here there is a simple union or synostosis of the arches and articular processes, the ribs at the same time being ankylosed to the spine. We had a case in the hospital (which Dr. Fagge described) of a young man who for eight years had had his back growing stiff until he was quite unable to stoop. He was seen to have his back arched, his neck stiff, and his thorax rigid. After his death from pulmonary disease his spine was found in the condition above mentioned, and the ribs also ankylosed to it. The specimen is in the Museum.

*Hæmarthrosis.*—We have already spoken of the occasional presence of blood in gouty joints. It has also been frequently observed in *hæmophilia.* In a case described by Dr. Wickham Legg fresh dark blood was found in the ankle-joint. In one knee there was brown staining of the synovial membrane, and in the other the tissues were of a dark colour, and there was also some commencing ulceration of the cartilage.

**Adventitious Growths and Loose Cartilages.**—Actively growing tumours are not found in the joints. When such tumours affect the articular ends of bone the joints are generally preserved by the resistance of the articular cartilage. Only one example, given in ‘Virchow's Archiv,’ do we know of malignant tumour spreading along the synovial membrane; it occurred around the knee-joint. But we have already stated that the joints in chronic rheumatic arthritis present pendulous bodies hanging into their interior, often in large number. This is especially frequent in the knee-joint, which is the favourite place for loose cartilages. A more local process of the same kind may cause the production of only one or two such bodies, which may then grow to a considerable size and become detached; thus it is that loose cartilages are probably formed, of which we have several specimens. One is as large as a florin, while others are smaller. They are flat, white, and smooth on the surface, although not even, having numerous depressions. Their structure is of fibro-cartilage, the cartilaginous element being in small proportion, or altogether absent in some; in others the structure is almost wholly cartilaginous, while in yet others there is some bone present; sometimes the apparent bone is only calcified cartilage, but in some examples there is true bone, with characteristic lacunæ and, perhaps, Haversian systems. The origin of these loose bodies has been the theme of much debate. It is generally agreed that the majority of them take their rise, as we just now said, from a localised process of the same nature as that observed in chronic rheumatic arthritis, that is, from tufts of synovial membrane which grow and then become pedunculated and cartilaginous. But some arise as fragments separated from the ends of the bones. Thus Mr. Poland removed from the knee-joint of a man who, in the dark, had struck his knee slightly against a corner of his bedstead, a piece of bone, that proved when the man died a few days after the operation to be a part of the patella; such an occurrence may easily be supposed to happen, though rarely. Sir James Paget also had a case where it was clear that a piece of the cartilage had been knocked off. But we have more than once met in the knee-joint with grains of various sizes and of a consistence like soft boiled rice, resembling a good deal the "melon-seed" bodies in the sheaths of tendons. These appeared to be on the road to the formation of loose cartilages, but much change was required to convert them into cartilage or bone.

**Degenerative Changes in Cartilage.**—*Degenerative erosion.*—Cartilage, which resembles the elastic coats of arteries in being evascular, also agrees with them in early showing a deficient power of endurance. A man has generally not reached his thirty-fifth year before his cartilages, like his aorta, show signs of being the worse for wear. The parts which reveal the change are those most pressed upon in use, such as the prominences of the condyles of the femur and the corresponding parts of the tibia; the patella has also a very special liability. It has been stated that when the leg has long been extended, as when confined in splints, ulceration of the cartilages of the knee will sometimes occur. A very good description of the anatomical characters of degenerative changes was given by the late Mr. Alex. Bruce, and our experience fully corresponds with that on which he based his statements. To the naked eye the earliest sign of the change is a yellowish spot, which afterwards becomes soft and flocculent and wastes into a cavity. Sometimes it is more yellow, and sometimes more fibrous, and sometimes more excavated. These varieties of appearances are, however, usually successive stages of the change. The flocculent surface is best brought out in viewing the cartilage under water. Such a patch very slowly spreads. Its appearance is liable to give rise to a sense of grating in the movements of the joint when the surface is exceptionally dry, but, as Mr. Redfern long ago pointed out, this change may make no sign of its existence. Thus to give one example out of many, a bricklayer's labourer, of thirty-five years of age, suffered a severe accident, crushing his leg, so that amputation of the thigh was required immediately after his admission to Guy's; we found the cartilage of the knee-joint much worn by this erosive process. On questioning the man he declared that he had never experienced the slightest sign of disorder in the knee; his occupation consisted usually of carrying hods of bricks up a ladder, and he had no idea that his lost knee had any fault about it; it had, indeed, always served him so well that the poor fellow would hear nothing against it. Microscopic examination of the cartilage under these circumstances shows the inactivity of the change. It is true that some multiplication of the nuclei of the cartilage cells may generally be seen, but we think too much has been made of this part of the proceeding. The most striking change is the fibrillation of the interstitial substance and separation of its normally homogeneous mass into bundles of the fibres so arising, while the cells do not show at all in these fibres, but appear to have very early burst and discharged their contents or to have been wasted away. This change is usually described as proper to rheumatic arthritis, but we think it is a general process, the result of repeated irritations and imperfect recovery through insufficient blood supply.

Sometimes the cartilage when so affected is said to be ulcerated, and we have often seen inexperienced persons misled by it when examining joints in rheumatic fever. Finding acute synovitis, with liquid exudation and lymph in the joints, associated with this degenerative change, they set all down alike to the rheumatic fever, and describe this as having caused "ulceration of the cartilages;" but this is certainly an error. The change found in such cases is exactly the usual erosion on the worn parts, and corresponds always in its extent with the elderly age of the subject; also, when other joints not inflamed in the same case are examined, they present the same erosion.

When considered on principles of general pathology the change in question shows successive stages, that is, in the irritative multiplication of the cartilage cells and the subsequent disintegration of the tissue. These same proceedings are found in ulceration of other textures, but the behaviour of the vessels in vascular parts is, of course, omitted in cartilage. Now, the active characters of an ulcer, such as we see on the skin or mucous membranes, are almost altogether dependent on the vessels, which swell and become choked with corpuscles, so as to induce death of the parenchyma dependent on them, while white and, perhaps, red corpuscles exude from their walls. The course of the ulcer, especially its healing, depends on the production of new granulation matter, containing new loops of vessel which the vascular tissue supplies. A tissue wanting in vessels and in the acts done by vessels wants also the characters by which all that we mean by an ulcer is substantiated, and it is more convenient to call the change in the non-vascular tissue by another name. The name "degenerative erosion" will serve well. For, although it has an active stage, and its cause appears to be direct irritation in the course of the use of the joint, nevertheless its leading characters are the failure of repair, and the change to a lower or less special type of fibrous tissue, which constitute degeneration. After acute ulceration of cartilage repair may take place, and therefore we occasionally find, on the examination of joints, the remains of healed ulcers.

Although the cartilage is dependent on surrounding structures for its vascular supply, yet it does not follow that any obvious change in these accompany its degeneration; for cartilage has its own proper self-nourishing power, and it is to the irritation and exhaustion of this that we must look for the explanation of its failure to maintain its integrity. Cartilage, like other textures, varies in its self-maintaining faculty in different people, so that in some it is sooner overpowered, just as in some people the head sooner becomes bald and the mouth toothless save for the dentist's assistance earlier than in others. Such failures, due to original weakness of constitution, even though that weakness leads to some irritative phenomena, are really degenerations.

We have already said that in many cases of caries of the spine the disease begins in the *intervertebral substance.* Softening may be constantly observed in persons who have been long bedridden. By thrusting the point of a scalpel in between the vertebræ the elasticity is found to be lost, and, if microscopically examined, the cells are found to have undergone a fatty degeneration. The fibrous tissue is seen dotted with granules, and the cells between filled with fat-globules.

**Eburnation of Bone; Ivory or Porcellanous Change.**—Like the last-mentioned change, this is sometimes described as proper to chronic rheumatic arthritis; but we often meet with it in joints which are otherwise normal, and hence regard it as of the same nature as the degenerative erosion of cartilage to which it usually succeeds. The bone exposed, when the cartilage is removed, is hardened and polished on its surface by the friction in the joint. Such surfaces have been spoken of as eburnated cartilage, but there is no trace of cartilage to be seen in them. They are made of indurated bone, in which microscopic examination reveals no structure, or only traces of it, so proving that mere mechanical pressure is the cause of the hardness, and that the layer should not be described as a development of a new bony surface. It is true that this change is most largely developed in chronic rheumatic arthritis, but it is not limited to this; it is sometimes found after carious disease of the joints, and often when there is no other sign of imperfection in the joints.

**Injury.**—Traumatic arthritis is constantly treated by the surgeon; the joint heals favorably under judicious management, though often the result is less fortunate. We have already spoken of the effects of acute arthritis, and alluded to the peculiar behaviour of the cartilage. In wounds of the joints the synovial membrane closes by a tissue resembling its own structure; but cartilage is not replaced by new tissue of the same kind, but by one simply fibrous. It will be seen in two or three specimens of fissures in the joints that fibrous tissue unites the broken cartilage. Mr. Poland drew attention to the curious fact that in the knee-joint the bony part of the patella may suffer compound fracture down to the cartilage and yet the cartilage remain unbroken. While speaking of injuries to the cartilage we may mention that in fractures of *costal cartilage* the union is never by new cartilage but by fibrous tissue, which, as the patient advances in life, may ossify. Such injuries are rare, but the few instances in Museums exemplify union by bone. The same occurs in the *laryngeal cartilage*—a fibrous union with occasional subsequent ossification.

# DISEASES OF BURSÆ, TENDONS, AND MUSCLE

## BURSÆ

**Bursæ** are serous sacs which form over bony prominences upon which great pressure is exerted; thus, a common situation for them is over the tuberosities of the ischia, or over the vertebra prominens or the acromion in porters, or more especially over the patellæ in those who work in a kneeling posture. Other bursæ are more deeply seated between great tendons and the bones they move upon. Any of these bursæ may inflame through excess of the pressure which caused them, or else, perhaps, through some rheumatoid tendency, and thus a swelling may form in one of the situations mentioned or beneath the deltoid muscle; or about the hip-joint, over the great trochanter, or under the psoas, or under the gluteus maximus; or about the knee-joint, under the extensor tendons, or around the semi-membranosus muscle, or under the ligamentum patellæ. These sacs undergo similar morbid changes to those already spoken of as occurring in the joints, but not in so regular a manner, seeing that their lining membrane is not so perfect. The sac may become filled with a large quantity of serum, or by a chronic inflammatory process a lymph may be thrown out which organizes until the whole sac is converted into a solid tumour; although the centre is generally then filled with a soft lymph or serous fluid. Sometimes, by a similar process, the lymph may form bands, which may be seen passing across the bursa, and become at last like hard tendinous cords; or its inside may be covered with shaggy tufts or pedunculated bodies, as seen in the joints, or filled with melon-seed bodies.

## SHEATHS OF TENDONS

**Inflammation.**—The sheaths of tendons are liable to the same affections as the joints; they may inflame and produce the various ordinary inflammatory products. We have already remarked that the sheaths of adjacent tendons may share the acute inflammation of the wrist or other joints, when severe rheumatic, or more especially when pyæmic, inflammation has occurred in those joints. Under these circumstances we have seen several of the sheaths filled with lymph or pus. An acute inflammation of the sheath with its enclosed tendon sometimes follows severe strains or punctured wounds, &c. The sheath then becomes filled with lymph, while the parts around swell greatly and the tendon may slough if the case be severe. The "thecal abscess" that thus arises may prove a formidable disease, bringing with it the risk of purulent absorption, and pyæmia.

**Ganglion.**—By a slower process a large quantity of synovia or serum may collect within the sheath of a tendon, and thus a pouch of fluid may form a "ganglion." Such circumscribed cysts may be distinguished as *simple or localised ganglion;* they are most common about the back of the wrist or on the tarsus, and being limited in size are not generally of serious consequence. But sometimes the great synovial sheaths about the flexor tendons of the hand or foot undergo a chronic inflammation and so form *compound or diffused ganglion;* the character of the inflammation then resembles very much that seen on the synovial surface in chronic rheumatic arthritis; thus, the interior of the sheath becomes shaggy with pedunculated growth and fringes, like those we have described as occurring in the joints, and if these become detached they fill the ganglion with hundreds of small bodies like melon seeds.

## TENDONS

**Inflammation.**—Besides the acute inflammatory affection which they share with their sheaths the great tendons are subject to a chronic inflammation, which is well known clinically under the name of rheumatism. But this chronic inflammatory state does not, so far as we are aware, produce any anatomical change beyond slight thickening. The crackling sensation which is met with in such cases appears to be due to the gelatinous effusion in the areolar meshes which we commonly find about such inflamed tendons. In *injury* the toughness of tendons is exhibited by their often resisting the most violent force, so that they separate at the attachment to the muscle rather than break across; thus, the quadriceps femoris will tear from its tendon more or less completely, and you will see in some specimens how, when the fingers were torn off, their tendons were drawn quite out of the arm. Chemical and vital changes they equally resist, so that in burns or sloughing wounds, or in the removal of a part by gangrene, you still see the tendons in the dead portion comparatively little altered. Those most apt to rupture from violence are the long tendons of the biceps, the ligamentum patellæ, and tendo Achillis.

**Repair.**—After tenotomy the cut ends of the tendon are drawn apart to some distance depending on the length of the contractile fibres of its muscle; this distance may be as much as two inches in the tendo Achillis, for the gastrocnemius naturally traverses a large space, crossing, as it does, two joints and having a large action on both. The interval is soon filled up by a soft material, which is formed by the surrounding parts, especially the sheath of the tendon which becomes congested at the same time. The tendon itself is almost evascular, and takes little share in its own repair, its part consisting in a swelling of the cut ends and a production of lines of new material between its fibres in continuity with that in the breach, by which means the connection between the ends is made firm. In a specimen of divided tendo Achillis we found a soft material joining together the divided ends twelve days after the operation, the microscope showed well-formed nucleated fibres lying together in the bundles. Mr. W. Adams has seen capillary vessels in the new matter about the eighteenth day, and in three or four weeks the required length of new tendon is formed, though yet not very tough. For a long time, if not permanently, the new part differs from the old in being less lustrous, and also in having a union with parts around which prevents it from retracting so much a second time if it is again divided, but it is composed of strong fibrous tissue like the original tendon, of which it well serves all the proper purposes.

**Ossification.**—This is occasionally observed in tendons; we have met with an example of sesamoid ossification of both heads of the gastrocnemius, such as that mentioned by Lobstein and others, and quoted by Virchow. The last-named learned author gives a full list of the seats in which these ossifications have been seen, as follows:—In the triceps and biceps brachii, digastricus (this also we have seen), rectus abdominis, adductor magnus, and diaphragm. But the most important examples are those called the *rider's bone* and the *drill bone;* the former appears in the form of a rough growth of bone, continuous with, or attached by ligament to, the outer face of the pubis or ischium, among the fibres of the adductors. Mr. Bryant gives a case in which he witnessed the formation of such a mass in an habitual rider. Drill bones were especially observed by Hasse in Prussian recruits, eighteen out of six hundred of whom had a growth of bone of variable size in the tendon of the deltoid from frequent injury of this part by rude contact of the rifle during drill.

**Club-foot.**—We shall only allude cursorily to the subject of club-foot, for it is fully described in the surgical lectures. Essentially it is a nervous disease, and the morbid anatomy of the nervous change which induces it is unknown. It is, however, occasionally associated with spina bifida; it is frequently congenital; it also frequently supervenes during early or even adult life, through paralytic or even spasmodic affections, sometimes following severe fevers, sometimes coming on through partial paralysis.

Club-foot arises from a loss of that balance between the several sets of muscles around the joint which preserves the joint in the position suited to its function. The balance may be lost through either spasmodic over action, or paralytic under action of either set of muscles. Which of these it is due to, in general, is a question that has been a great deal discussed, because it affects the indications for treatment very obviously. But we must not rest too certain that in all cases there is such a loss of balance of muscles. For when all the muscles of a joint equally put forth their power on it, the result is not always that the joint is in a neutral posture, but it may be bent in a constant direction. Thus, in *rigor mortis* the position of the foot is one that, if permanent, would constitute a slight amount of talipes varus and equinus; the heel being drawn up, and the sole of the foot turned inwards. If such a condition of equal action of all the muscles supervened during life, the effect would be to induce talipes equino-varus, although no set of muscles was either paralysed or in greater spasm than the rest; the position being the result of the natural preponderance of the power of one set of muscles over the others. It is to be noted that the most frequent kind of congenital club-foot is varus; the most common kind of acquired talipes is equinus; while talipes valgus is a rare affection.

**New Growths.**—Tumours sometimes spring from tendons, but such an origin of them is not common. We have several examples of *carcinomatous* tumour, one arising from the sheath of the tendon in a finger; another from the tendons in the palm of the hand; another from the biceps femoris, &c. We have also some specimens of *sarcoma* arising from aponeuroses, and an *enchondroma* which appears to grow from the extensor tendons. In most cases, however, tumours on the tendons are found to be of the nature of *indurated bursæ.*

## MUSCLE

**Malformation.**—Extra muscles occur under certain conditions, and are pointed out by anatomists; or muscles may be sometimes absent, as in a case which occurred here several years ago, where the pectoral and adjacent muscles were wanting.

**Injury.**—When a muscle is injured or destroyed it is repaired with a fibrous structure, and never by the reproduction of muscular tissue; if such a muscle be put to much use the fibrous structure assumes the consistence of ligament. *Spontaneous rupture* may occur from violent spasm, as in tetanus, of which Mr. Curling has given many examples. We have seen the rectus abdominis thus torn; but rupture in tetanus is more common, we think, in the muscles of the back, where we often find laceration and effusion of blood. Rupture of muscle occurs also during violent effort in delirious persons, or from accidental injury suffered by them in unguarded movements made during delirium. This is, no doubt, the cause of some of the ruptures of muscle which we find in the bodies of persons dead of fever; but the changes we shall presently have to describe as occurring in the muscles in typhoid may have a share in causing the ruptures. We have met with suppuration of a ruptured rectus in typhoid, an abscess being formed between the ends, where the muscle was completely torn across and showed abrupt ragged edges. In the cases we have seen, the rupture has usually been through the fleshy part of the muscle; but we have twice known the extensors of the thigh partly torn from their tendon. The other muscles which have been known to rupture are the biceps femoris and gastrocnemius.

**Inflammation.**—This, when attended with the usual results, as suppuration, gangrene, &c., is by far most frequently pyæmic, or due to thecal abscess, or other neighbouring active disease; but the muscles will suppurate spontaneously. Thus, we have sometimes met with psoas abscess where only the muscle was affected, and we have had two or three examples of great abscess in the deep gluteal region extending into the pelvis through the ischiatic notch when the bone was not affected, and the muscles were the parts principally destroyed; we meet with them also in the abdominal walls. Suppuration arises and proceeds in the cellular tissue among the fibres, and the pus-cells may be seen in lines between the muscular fibres before these are themselves involved. Myositis is seldom seen in the acute stage; if so, the colour of the muscle is lost and an exudation has taken place into the substance of the muscle. The microscope shows the fibrillæ granular and leucocytes abundant. It generally arises from injury which, if not ending in suppuration, terminates by production of fibrous tissue. This is its mode of repair. The effects of chronic inflammation we shall presently speak of under degeneration.

**Hypertrophy.**—*Hypertrophic paralysis.*—Hypertrophy of voluntary muscles does not become a morbid condition. When well pronounced, as in the blacksmith's arm, it does but show what size the full nourishment of the muscle will bring it up to. A very interesting condition of muscles has been pointed out by Duchenne, of Boulogne, under the name of pseudo-hypertrophic paralysis. It is found generally in young boys. In the cases we have seen there was general, incomplete, muscular paralysis, affecting, indeed, all the muscles, and associated with the usual wasting of most of them; but some of the muscles, especially the gastrocnemius, glutei, and erector spinæ muscles, instead of wasting, had remained large, or had even increased in size beyond their normal volume, while they were as much paralysed as the others. When examined under the microscope the muscular fibres are found much wasted, and amongst the atrophied fibrils a production of granules, fibre, and fat. The presence of the latter is the most striking object observed.

**Degeneration and Atrophy.**—These are usually chronic processes, and are generally of the fatty, granular, or fibrous kind, though pigmentary degeneration sometimes occurs in the heart, and a waxy degeneration is spoken of in progressive muscular atrophy, which, whatever its nature, must not be confounded with the waxy lardaceous change in the liver, and other viscera.

*Fatty degeneration.*—This degeneration shows itself as a deposit of fat-grains within the muscular fibres. It is usually a chronic process, but we have seen an acute fatty degeneration in a case of poisoning by phosphorus, the muscular fibres not only of the heart but also of the voluntary muscles being very much charged with fat-grains, so that the transverse striæ in many could not be seen, and this although life was not prolonged more than eight days after the poisoning. Others have made the same observation. This acute fatty degeneration, or *steatosis* as it has been called, implicates also the liver and kidneys; in arsenical poisoning we have verified its occasional occurrence; it has also been found in poisoning by antimony as well as in acute atrophy of the liver. In all of these conditions the disorder is of short duration. Such rapid development of a change once thought to be always chronic is a suggestive fact. The fatty muscle to the naked eye is soft, pale, or yellowish. The microscope shows absence of striæ and presence of fat-globules. The nature of the latter is also proved by the action of osmic acid, which makes them black.

The production of fat-grains in the fibres of voluntary muscles is not so frequent as in the muscle of the heart, where it is so often noticed. The proportion of fat present when this change is microscopically well marked is very small, not more than 5·7 per cent.; and, indeed, this granular fatty change of the fibres never much alters the red colour of a muscle, at least, of voluntary muscle.

But we often see an evident fatty change in the voluntary muscles of old people, particularly of those long bedridden, or in the bodies of old paupers in the dissecting-room who had long fed on workhouse diet. In such bodies we may see the whole muscle, say a pectoral, apparently almost changed to fat; in another case streaked with this fatty change; or, yet more frequently, we see this fatty change in the long disused muscles around an old scrofulous hip- or knee-joint. A fatty degeneration of the diaphragm is often an important condition to notice. The microscope will show the nature of this change, and that it is a totally different condition to the granular degeneration we have been speaking of. What looks like fat is really histological fat, the proper cells of which are found insinuating themselves between the muscle-fibres, at first chiefly in the course of the vessels, afterwards in all parts, so that they waste away, a wasting which the disease that induced the formation of the fat much encouraged, and, perhaps, indeed chiefly caused. But disease is not the sole cause of this fatness of muscle, for it is habitual in over-fattened animals. The wasting of the muscular fibres under these circumstances is generally simple and not accompanied by any very marked granular change.

*Fibrous or fibroid degeneration.*—This is also often found in the neighbourhood of chronic inflammation, hence it is termed myositis by some authors. Portions of muscles around carious joints, which in other parts show the fatty state above described, are often seen as a hard white structure in which the unaided eye perceives the remains of muscular fibres visible by their linear arrangement. This fibrous change is the natural result of chronic inflammation in muscle, and is found after repeated rheumatic attacks and after syphilitic inflammation. The change is best known in the heart. The muscle is converted into a tough semitranslucent white substance, which, when the change is complete, is found microscopically to be well-formed fibrous tissue. The earlier stages are not well known. They are best studied about old knee-joints, and near periosteal inflammation in the deep muscles of the thigh and leg. The new material follows the vessels, so that a corpuscular new formation surrounds them. We never could decide whether the corpuscles arose through exudation or through multiplication. But as to the permanent new fibrous tissue we believe it to take its form from the pre-existing fibrous texture as, indeed, all scar-tissue does, while the new corpuscles of the inflammatory act are probably derived from the vessels, and do not take part in the healing process that forms the fibrous scar.

A remarkable *induration of the sterno-mastoid muscle* has long been observed in infants, and its causes have given rise to much controversy. There are those who have regarded it as syphilitic when there have been suspicions of the existence of this disease, whereas others have regarded it as the result of injury. Dr. S. Taylor had an opportunity of examining a case after death, and he found the muscle infiltrated with fibroid tissue, and Dr. Goodhart had another of the same kind. It has been suggested that it may have been due to violence during labour by traction on the head. There is no proof of this, but hæmatoma of the sterno-mastoid has been observed in new-born children as if caused by torsion of the neck.

Besides such fibrous degenerations, which are probably inflammatory in their origin, there are others that are certainly passive. Thus, the over-stretched columnæ carneæ of a dilated left ventricle become changed to fibrous tissue.

In progressive muscular atrophy, or Cruveilhier's paralysis, the muscles waste away because of a degeneration of cells of the anterior grey horns of the spinal cord. The tissue of the muscle in the several cases we have examined has shown little more than a simple wasting. The electrical excitability, you will remember, persists in this disease as long as any fibres of the muscle are preserved. Dr. Lockhart Clarke describes a fatty, fibrous, and waxy degeneration of the muscle in this disease ; others also describe a fatty change. We would not deny that some fibres may be found so affected, but it is remarkable how simple the atrophic process commonly is, the fibres growing smaller and smaller, still preserving their striæ until the then remaining parts break into yellowish grains.

In muscular wasting through other paralytic nervous diseases the same is true, as far as we have been able to see; the wasting is a simple dwindling with little alteration of structure. We have not been able to satisfy ourselves of the irritative proliferation of the sacrolemma described by Erb as occurring in muscles whose nerves are artificially destroyed, and by Mannkopf in muscles wasted in acute myelitis. These authors are quoted by Charcot to prove that such signs of inflammatory irritation in the muscles accompany the loss of electrical contractility in irritative diseases of the spinal cord and nerves.

*Myositis ossificans.*—We have spoken of ossification of the tendons, but ossification of muscle is rare though sometimes met with. Messrs. Hutchinson and Godlee exhibited at the Pathological Society cases of young boys in whom this had occurred. Large plates of bone existed in the erector spinæ, and more especially in rhomboid, trapezius, and latissimus dorsi, which prevented them stooping. One patient had suffered from an obscure illness some time before these were noticed, and it was thought that a myositis might then have occurred.

**Waxy Degeneration (Myositis Typhosa).**—We have already mentioned the occurrence of abscesses about ruptured muscles in fever. Abscesses have been long known to occur in fever, and were looked upon as part of a pyæmic process until Zenker drew attention to certain changes which parts of muscles sometimes undergo in typhoid. This condition has received much attention in Germany, but little has been discovered in England concerning it; we have not been fortunate in meeting with examples of it, unless some incipient abscesses, one in rectus femoris, one in rectus abdominis, which we should have ascribed to injury, were really of this nature. It is not peculiar to typhoid, but appears also in cholera, acute tuberculosis, smallpox, and other diseases.

The change is said to affect chiefly the adductors of the thigh, the abdominal and pectoral muscles, and the diaphragm, as a circumscribed patch from one to three inches in diameter, in which the fibres are seen swollen and waxy, or so softened into a reddish pulp that the whole looks like a muscular abscess. The appearances presented microscopically are those of acute inflammation; the sarcous substance swells, and loses its striation, becoming translucent, as if from fusion of its sarcous elements; inflammatory corpuscles meanwhile appear between and within the fibres. Rindfleisch says these cells show a peculiarity in their large size, which allies them to the proper cells of "typhoid deposit" in the intestine. After a time the old fibres waste away, and new ones appear inside the sarcolemma tubes in the form of long multinucleated spindles, such as are found in the original development of muscle.

It would be interesting to inquire whether the phlegmasia dolens, which occasionally follows typhoid, has any connection with this myositis typhosa in the adductors of the thigh.

**Tubercle.**—The occurrence of tubercle in muscle is very rare, and we have no specimen to exhibit.

**Syphilitic Disease.**—This, on the other hand, is very important, and not very infrequent in the muscles. It might easily be mistaken for tubercle when in the caseous form, with softening in the centre. Syphilitic inflammation of muscle is found showing characters according to the degree of intensity and the stage of inflammation, as well as the extent of structure implicated. It is only rarely now, since Bouchier drew attention to their nature, that we meet with examples of syphilitic formations removed by excision from the muscles of the limbs; such occurrences were formerly by no means rare. When so removed we have seen them in the form of *caseous* masses; one was as large as a hen's egg, the interior softened down. The microscopic structure of caseous syphilitic myositis shows the usual characters of syphilitic disease, *i. e.* a highly corpusculated lymph, whose elements resemble those of ordinary granulation, and tend rapidly to decay. Sometimes instead of a cheesy mass we find a syphilitic *fibrous* tumour, not quite like other fibrous tumours, which are circumscribed, and displace the muscle in their growth; but rather formed between the muscular fibres out of a lymph developed interstitially and from the perimysium, so that they may often be seen to contain the relics of the atrophied muscular fibre within them. These syphilitic tumours are found more often in the tongue, and in the muscles of the legs and forearm. We have seen them in the sterno-mastoid muscle, and have examined a specimen removed from the deep muscles of the thigh. Besides these *circumscribed* masses, which would, at first sight, be called tumours, syphilitic myositis appears in a *diffused* form both in the heart and in the muscles of the limbs. Indeed, it is most important to be well acquainted with the effects of syphilis on the muscles of the limbs. We have met with cases which had been called progressive muscular atrophy, and, indeed, closely resembled this terrible disease, yet which rapidly got well under iodide of potassium in spite of the grave prognosis which had been given. Such cases of general wasting, however, we think, are due to syphilitic nervous rather than muscular disease. We have several times seen the diaphragm involved in syphilitic disease, when the latter has attacked the liver or spleen.

**Morbid Growths.**—*Cancer.*—The muscles are frequently invaded by malignant tumours arising in the breast or the bones. Also in the lips and the tongue the spread of epithelial cancer from the mucous membrane or skin into the muscle is frequently enough met; but *primary* tumour of muscle is rare. Mr. Teevan has carefully collected many cases of primary cancer of muscle, of which nearly all were called medullary cancer. The two exceptions called scirrhus are doubtful. Of the few cases we have seen one in the right rectus abdominis was medullary sarcoma. The term medullary cancer, as used in the description quoted by Mr. Teevan, would not be distinctive between different kinds of rapidly growing malignant tumours, which are all apt to be called medullary cancer whether they be sarcoma, lymphoma, or carcinoma. From a practical point of view this is of no importance whatever, and, indeed, the lines of distinction between the forms of growth we have named reach their vanishing point in the vague histology of such tumours as these soft, swiftly-formed, malignant cancers of muscle, whose structure does not offer particulars enough for useful distinction.

It is remarkable that all the cases Mr. Teevan collected occurred in the larger and more superficial muscles. This, however, may only be because tumours in deep and small muscles would not have been sufficiently recognisable and removable at the stage when they are limited to the seat of their origin.

When cancer does invade muscle or arise in muscle, its spread in it is generally free. In the cases we have examined from the neighbourhood of cancers in breast the growth has been found extending between the muscular fibres. What ultimately becomes of the fibres themselves it is very difficult to see. We do not think it can be confidently said that the proper sarcous substance has no part in the formation of cancer. It is very certain that if the muscles be examined around a spreading cancerous tumour, nucleated cells may be found in parts which are apparently healthy to the naked eye, and these are always appreciable with facility, but more especially in the tongue can they be discerned, because the cells and nuclei are of large size, and often contained in compound cells, the cancer being of that kind which is called epithelial. In such a case very often, if the muscle be examined for some distance around the disease, these large cells may be met with in the course of the fibres, a microscopic sketch of which we have by us. For we have seen the muscular fibres, though at first resisting the growth and merely compressed by it, at length, that is at a greater depth, appear to divide up into spindle-like bodies, each with a nucleus behaving, indeed, like the hepatic tissue behaves in presenting intermediate stages of transformation from the normal elements to cancer-cells. In epithelial carcinoma of the tongue we have convinced ourselves of the growth of cancer-cells within the muscular fibres. This is often well seen. It has been insisted on by Weber, the accuracy of whose drawings we can fully verify. It has been urged by some that the pressure of the cells indenting the fibres would produce the appearance of their entry into it; but the appearance of cancer-cells within the fibres is so frequent that this explanation will not hold good.

It is important to notice the free spread of cancer in muscular tissue. We recently examined the line of section of a cancer removed by galvanic cautery from the tongue, and found cancer-cells present close up to the surface of excision, so that no doubt could be entertained that cancer-cells had been left, though the part had to the naked eye all the appearance of being perfectly healthy. The practical importance of this is evident. It is necessary to remove a muscle largely around any cancerous spot within it. Mr. Teevan urges the necessity of always taking away the whole of any muscle that has been invaded by cancer, and, no doubt, this is a good rule so far as it is practicable.

**Fibrous Tumours, &c.**—The other primary tumours in Mr. Teevan's list were generally described as fibrous; we have examined such tumours; one from the semi-membranosus was a fibro-sarcoma, the fibrous element predominating. There were no other tumours in the body. No case of *lipoma* is recorded, and other forms of tumour are rare; but Mr. Teevan collects five examples of erectile (cavernous) tumour in muscle. *Cysts* are not often met with in muscles. In one we lately saw the origin was probably a softening of one of the fibroid masses just spoken of.

**Parasites**—*Trichina spiralis.*—The entozoon peculiar to the muscles is *Trichina spiralis.* The very old specimen, preserved in the Museum, contains many. But the nature of the affection was unknown at the time when it was put into our collection. It appears that Mr. Hilton first suspected that the little grains might be parasites, and Professor Owen recognised them as worms, and called them with the name by which they still are called.

Dr. Hodgkin, in his 'Lectures on Pathology,' says, "The first observation of these little bodies with which I am acquainted was made in 1828 by my friend Mr. Peacock, who considering it a remarkable affection of muscular structure made a dry preparation of the omohyoid to exhibit it. In this state the true nature of the affection was neither known nor suspected. A short time after, my friend John Hilton observed two or three cases in the dissecting-room at Guy's, and drew up a good account of its occurrence and sent this to the Med.-Chir. Soc., Jan. 22nd, 1833, entitled, 'Notes of a Peculiar Appearance in Human Muscle, probably depending on the Formation of very small Cysticerci.' The paper was read but the publication suppressed by the Council.* About two years after, the nature of this formation was further investigated by Mr. Paget and Mr. Owen, who gave it its name."†

Trichina spiralis is very infrequent in this country. In our own experience we have not met with it oftener than once in five hundred bodies examined by us, if so often as this. The affection is known by the voluntary muscles appearing to be marked throughout with countless white specks. When more closely examined the whole tissue is seen to be pervaded with little white oval bodies; they are hard, and always lie with their long axis in the direction of the fibres; they are placed at regular intervals not crowded into masses. If picked out and placed under the microscope they resemble miniature eggs; the shells or cysts are composed of earthy matter and are very hard, requiring some force to crush them. When one is broken carefully a round worm emerges; it is pointed at the ends, especially the caudal end, and it is the encysted larva of a small nematoid which would reach its sexual perfection in intestines of flesh-eating animals. These creatures are distributed in all the muscles with striated fibre, except the heart. Virchow is said to have seen them in the heart, but the usual rule is shown in our preparations, where the heart is free, and yet the pharynx has its involuntary striated muscle full of them, like the biceps, which has been chosen here to represent the general muscular system, though muscles of the trunk would have served better, since they contain more trichinæ. When thus encysted, as they have hitherto been found in this country, there has been no history of any ill effects from them. It appears from the observations of continental pathologists, especially Virchow, Leuckart, and Davaine, that it is only when the worms are moving to take up their positions in the muscles that the serious and, perhaps, fatal mischief they cause is observed. The following is a short abstract of their history as given by the last-named author. The sexually mature worms inhabit the intestines of many kinds of animals; they very quickly reach their maturity on entering the intestines. They are viviparous, and their young, soon after birth, begin wandering through the intestinal wall, penetrating on their way the peritoneum, and working on until they reach the voluntary muscles in whose interstitial connective they must lie. Their progress excites a traumatic irritation, which locally produces a granular change in the fibres with serous effusions, pains, and paralysis; besides this they give rise to exhausting fever and, perhaps, peritonitis, from either of which the sufferer may die.

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* 'Med. Gaz.,' vol. xi.

† 'Trans. of Zoological Society,' 1835, and Dr. Farre's paper, 'Med. Gazette,' Dec., 1835.

The symptoms are in proportion to the number of the creatures. In their position in the muscular flesh they wait the time when their host may be eaten, and it appears that in that larval state they are very tenacious of life, though they are very perishable when adult.

*Cysticercus* (*telæ*) *cellulosæ.*—This entozoon is the cystic worm whose presence constitutes "measles" in pork. Though not frequent, it is far from very rare in man. Its more usual place is the cellular tissue of the muscles; but it is found in other parts. Thus, Griesinger collected more than fifty examples of cysticercus in the brain. It is generally of small size, not larger than a horsebean or hazel nut. It consists of the cystic caudal extremity of the animal with the head and neck coiled spirally and embedded in a spot on its surface.

The *Echinococcus*, or true hydatid, may be found in the muscle as in all other parts of the body.

# DISEASES OF THE HEART

## PERICARDIUM

**Malformation.**—The pericardial sac has occasionally been found altogether absent. Some of the old writers had already observed this condition, but discredit was thrown upon their statements on account of the very frequent obliteration of the pericardial cavity by adhesion, a fact which was thought a sufficient explanation of the apparent absence. This supposition might have been true in many instances; but, at the same time, such a want of development does sometimes occur; for, some years ago, we had an opportunity of seeing a specimen of the kind, brought to the Pathological Society by Dr. Baly, where the heart was uncovered, and lay in the cavity of the pleura against the left lung.

**Hydropericardium.**—The pericardial sac is liable to simple serous effusion like other serous cavities. The effusion is said once to have amounted to eight pints, but it is usually under half a pint, and then forms part of a general dropsy. A certain quantity of fluid is found in the pericardium in all cases when the body has lain some time after death. But the amount is not large, not more than an ounce or two. If more than this be found, its cause should be sought in some obstruction of the vessels; such an amount as six ounces, often quoted from Corvisart as possibly normal, would certainly be due to disease. Mr. Hilton proved it to be due, in some instances, to absence of the *valvula Thebesii*, which should guard the orifice of the coronary sinus. We certainly occasionally find the pericardium greatly distended with fluid without any apparent cause; perhaps most commonly in children, in whom the pericardial effusion is the only evidence of the "rheumatism" to which its presence is sometimes attributed.

**Inflammation**—*Acute.*—In ordinary acute pericarditis the earliest stage seen is a minute injection of its vessels, causing a blush of redness, which close observation resolves into a beautiful red network. This injection is almost a certain proof of pericarditis, and when present there will be found at the base of the heart, and about the great vessels, some shreds of inflammatory lymph. This earliest stage is soon over; in a day, or two at most, a fibrinous layer is formed, which, in the course of a few more days, forms a complete covering to the heart, and which, if there is no fluid exudation, may connect the surfaces of the heart and its sac rather firmly. This forms the second stage or degree of pericardial inflammation. We more frequently see some serous exudation mixed with the solid lymph, and thus the latter is found in shreds, or presenting a honeycomb appearance, very much as if a layer of butter had been placed in the sac and the surfaces suddenly separated; but when the quantity of fluid is small, one notices that the parts of the heart which are kept in contact with the pericardium show a surface like that of a file, with rugosities across the direction of the friction which has caused them; such roughness of the lymph gives rise to a rough, rasp-like, friction sound.

Very early pericarditis is generally met with as a complication of morbus Brightii, but rheumatic cases for the most part survive until the disease is more advanced, and then a thick layer of lymph will be found covering the heart; and if there be much fluid effused with it, an occurrence not usually met with until a considerably longer period has elapsed, and more proper to fatal rheumatism, then the more solid part puts on a very remarkable appearance. From the two serous surfaces not touching each other shreds of lymph float in the fluid, and the surface is, consequently, covered with long shaggy processes. This is the condition we generally have in our minds when we speak of acute pericardial effusion as it occurs in rheumatism.

Occasionally the inflammation, through either long duration or intensity, or else through the low state of the patient's powers, is purulent, and thus, in the same way as a pleurisy ends in an empyema, so may a purulent effusion result from a pericarditis; it is not very common, and no doubt is generally fatal. It usually owns a cause different from that of either of the fibrinous forms we have described; we have seen it once when an aneurysm had approached and compressed the pericardium. The bursting of hepatic or other abscesses into the pericardium may cause it; but it sometimes exists without any obvious or assignable cause. In one case, of which we have a drawing, the pericardial sac was immensely distended, and within it were thirty-six ounces of pure pus: the man had been ill two weeks.

In some severe cases of acute pericarditis, the heart's muscle beneath the pericardium is found to undergo a fatty change to a depth of one sixteenth to one eighth of an inch, the amount of fat in the muscle fibrils being so large that the layer becomes conspicuous to the naked eye as a yellow line under the pericardium.

If a section be made through the walls of the heart, together with the inflamed membranes, there will be seen most externally shaggy lymph, in which leucocytes and blood-corpuscles may be found; beneath this a granular layer of lymph, then a little natural adipose tissue, and, beneath all, the muscular fibre granular and fatty.

No doubt in very slight cases the new lymph will disintegrate, and complete recovery by resolution will be the fortunate result. Severer cases undergo simple adhesion of the opposed surfaces, with little or no bad consequences. In the graver cases the adhesion is usually complicated by the effects of inflammation on the heart, and is only one item in the consequent morbus cordis.

*Chronic.*—As we have already had occasion to mention, the so-called chronic inflammations are often altogether different in kind from the acute, being slow in their origin as well as progress, the process of production of new material being more allied to that of a growth than of an exudation which has subsequently organized. In the pericardium, however, most of such chronic changes have an acute origin; for example, after a pericarditis and effusion of plastic lymph a continued succession of exudatory layers takes place, and, if the case be fatal within a few weeks or months, the various stages of the process are seen. Thus there may be a firm layer of fibrin adherent to the heart, and over this a softer and more recent exudation. The heart's substance will be found more or less implicated; we have mentioned above the effect of acute pericarditis in causing acute fatty degeneration of the muscle beneath. In chronic cases we may find a growth of fat beneath the membrane, with or without an extension of fibrous tissue into the superficial layers of muscle.

Occasionally, however, though much more rarely than in the pleura or peritoneum, chronic changes may be met with which seem to have had no acute commencement. Thus the visceral pericardium is much thickened, but not adherent to the parietal layer, involving the heart in a fibrous layer, uneven on surface, and encroaching often on the muscular texture of the organ itself. The fibrous patches are found spread irregularly over the surface. From the absence of adhesion it is difficult to believe that true inflammation occurred in these cases; the change is exceedingly slow. We often find the fibrous tissue dipping into the underlying muscle of the heart.

**Adherent Pericardium**—*Simple.*—As a result of pericarditis the serous surfaces may become wholly or partially adherent, or the membrane merely thickened in particular spots. Thus, several years after an attack of pericarditis which has been cured, an adhesion may be found attaching the front or other part of the heart to the sac; not uncommonly the apex is thus adherent, and sometimes a long loose cord is seen attached to it, as of a band which had broken. More frequently than this the heart is found universally adherent to its serous covering by means of a most delicate areolar substance, and then no evil appears to result from it; thus we found it three times in one week on one occasion, and on another four times in one week, always innocent of any share in the fatal illness. We cannot, therefore, but think that its occurrence is quite unconnected with symptoms, or any impaired action of the organ. It might be supposed that, if nature has made the heart to move freely in a serous cavity, the obliteration of this would of necessity impair its motions; but the same might be thought of the lungs; these organs, in like manner, are intended to be free in the chest, and yet adhesions appear to do no harm, and are found, more or less, in a large proportion of the cases we examine. There may be, indeed, some amount of impairment of action in either case, but this is not appreciable, nor are any ill effects recognised; indeed, when we reflect that no vacuum arises in the pericardium during the action of the heart, so that the sac must follow the heart's movements, the delicate adhesions would afford no hindrance to its action. When such adhesions were first recognised (at the time of the discovery of auscultation), it was thought that an enlargement of the heart resulted therefrom, and no doubt the two conditions may often be found combined; but in many of these cases it will be seen that valvular disease is also present; in others the enlargement might be considered to be due to other causes, since a hypertrophy often takes place without any pericardial affection. Our own opinion is that simple pericardial adhesion produces no appreciable untoward consequences.

*Fibrous.*—But there is another class of cases, the result of graver pericardial inflammations, in which the adhesion of the pericardium is by a thick vascular fibrous layer, and the muscular substance is torn by the attempt to separate the pericardium from the heart. The heart is then always found dilated and hypertrophied, and there may or may not be valvular disease coexistent, for the disease is always the result of a severe, and often of a general, carditis. To understand how such cases come about, we must remember what the change is under the acutely inflamed pericardium which we have already mentioned. It is natural to expect that such a damage of muscle must lead to loss of power and temporary dilatation of the cavities, which dilatation, creating a greater mass of blood to be moved, furnishes that resistance which the heart is always so ready to respond to by hypertrophy, and in this way a grave pericarditis leads at last to the dilated hypertrophied condition we are speaking of.

As a consequence of this pericardial thickening all the usual symptoms of cardiac disease may be produced, and such cases constitute some of the most difficult of diagnosis with which we meet.* They too have often been aggravated by the extension of the pericardial inflammation to the mediastinum and to the cellular tissue of the neck, causing compression of the great veins.

*Calcareous.*—We sometimes find the heart enveloped in a bony case, so that it is difficult to understand how the rhythmic action of the heart could have been continued under such restraint. This calcareous ring may extend deep into the tissue of the ventricle, and is usually found at the base of the heart; sometimes smaller bony patches are met with on the anterior surface, and sometimes on the posterior surface towards the apex, causing adhesion to the diaphragm. These plates may result from the inflammatory processes before mentioned, but probably are usually degenerations occurring in the adventitious fibrous tissues. It is worthy of remark that bony plates exist naturally in the pericardium of some animals.

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* 'Guy's Hosp. Rep.,' Series 3, vol. xvi, p. 197.

**White Patches, or Milky Spots.**—The question has long been under discussion, whether the white patches so often seen on the surface of the heart are inflammatory or not; some looking upon them as mere thickening of the membrane, and others as the result of inflammation. If by inflammation we mean that there has been a pericarditis, and lymph effused, which has subsequently organized, there is no proof that such has ever occurred. The obscurity of the term inflammation has probably had much to do with the difficulty in determining the point, for no doubt most pathologists are assured that such patches are of slow formation. If this be granted, it is another question how far the term chronic inflammation can be applied to such a process. Occasionally, as a result of pericarditis, some of the lymph may remain and organize, producing thick white patches on various parts of the heart, such as we have already described; but these are mostly irregular, and at base or apex, while the white patches of which we speak are always on parts of the organ which come in contact with the sternum, the fifth and sixth ribs, or otherwise suffer most friction. Thus on the front aspect, in the middle of the right ventricle, a circumscribed white smooth patch may often be seen, also on the apex of the left ventricle in front; the same also on the posterior aspect, especially on the projecting coronary vessels, and sometimes on the right auricle. Hence we believe them to be due to *attrition*, and formed therefore in the same manner as callosities in other parts, such as corns on the toes; which are not, if of slow formation, generally called inflammatory. It has been stated that they are very frequently found on the hearts of soldiers, where they are thought to be due to the wearing of the cross-belt. It is much in favour of the view which regards them as due to attrition that they are generally found on large strong hearts, especially the hypertrophied hearts of granular kidney cases, though it might be as well to remark that we have twice met with a small layer of recent lymph limited to such patches; and that it is just in these cases of granular kidney and large heart that we are so apt to meet with acute pericarditis, which it might be suggested may be due to friction on the covering patches.

**Hæmorrhagic Effusion.**—It occasionally happens that, on opening the pericardial sac, blood is found within, and a question immediately arises as to its source. If it be pure and clotted, it has generally flowed from a burst aneurysm or ruptured heart, but if it be in small quantity, then, on closer inspection, we may find the serous surface covered with lymph, and it becomes apparent that the blood is merely an accident of pericarditis; in one or two cases lately, we have seen much importance attached to this presence of blood, and the vessel carefully looked for whence it was supposed to emanate; but it is merely an accident of the pericarditis, and depends, probably, upon some constitutional cause; for a disposition to purpura may often be observed elsewhere in the body, and hæmorrhagic pericarditis has been occasionally observed in an epidemic form, suggesting an association with scurvy. The source of the blood is either from new-formed vessels, which, being yet tender, have given way ; or, in the absence of proof of the existence of such, it is more likely that the vessels of the serous membrane have furnished the supply; these being highly congested, and the tissues softened. A similar sanguineous effusion is sometimes noticed in the pleura and peritoneum. Occasionally, where the lymph is solid, and the pericardial sac closely in contact with the heart, the blood is found in coagulated layers, interspersed with the white laminæ of lymph.

**Air in the Pericardium.**—Whether air can be secreted from a closed surface during life is a question much discussed by pathologists. There can be no doubt that in most cases air found in closed cavities of the body results from decomposition ; and with respect to the heart, we have never met with air in the pericardium, unless its source was clearly either putrefactive or an entry from without during life through a communication established with some air-containing cavity; as once with the œsophagus, or as in another case, with a hepatic abscess that also communicated with the intestines, or in a third with a phthisical vomica. Of secretion of air into the pericardium we have no experience. In one case, where, owing to very remarkable sounds, it was thought air might be present, the patient recovered.

**Morbid Growths and Adventitious Substances.**—*Fat.*—This often exists in excess under the pericardium, but its importance is connected generally with degeneration of the tissue of the heart with which it is associated, and therefore we shall refer to the subject hereafter.

*Cancer.*—In cases of widely spread cancerous deposits throughout the body the pericardium may also be included, and numerous white elevated patches of the deposit may be found upon it. Also, in cases of cancer about the root of the lung, malignant growths may penetrate the serous sac. They then are found in the form of nodules growing into the pericardium about the pulmonary veins. Their pressure may cause a direct disturbance of the heart's action and so add much to the whole sum of misery induced by that terrible disease.

It is remarkable to what extent the heart may be involved in a new growth. Thus Dr. Peacock took to the Pathological Society some years ago a specimen where the heart was completely embedded in a mass of cancer, and Dr. Broadbent had a case where the whole pericardium was converted into a tough, thick, sarcomatous growth.

*Tubercle.*—In the same way as cancer may occur on the serous sac, so may tubercles in cases of general tuberculosis. They are seldom, however, found without an inflammatory process being present at the same time, and thus the affection is analogous to a tuberculous pleurisy or peritonitis, with both of which the tuberculous pericarditis is sometimes associated when no other tubercle is found in the body, the whole forming a peculiar diathetic disease of the serous membranes to which sufficient attention has not been given. The three serous membranes often are in exactly the same state. The inflammation is essentially chronic, and so the disorder may be looked upon as one of the forms of chronic pericarditis. A good specimen is in the Museum, where the pericardium is closely adherent, and where on separating and tearing open the lymph, a quantity of tuberculous matter was seen amongst it.

Mr. Morrant Baker showed at the Pathological Society a most remarkable specimen taken from a man who died of renal disease. The heart was found surrounded by a layer of omentum, which came through a hole in the pericardium and diaphragm. Owing to the existence of a scar on the skin it was thought that the hole was due to a stab and was not congenital.

## SIZE AND FORM OF HEART

**Misplacement of the Heart.**—In cases of a general transposition of the organs of the body, the heart may be found on the right side, but sometimes quite independently the heart may be situated on the wrong side. We know the case of a little girl, aged three years, in whom since birth the impulse and sounds of the heart have been found on the right side corresponding exactly in position to their site on the left. One of the physicians of "l'Hospice des Enfans Trouvés" has met with four examples of this kind without any other organs being misplaced.

**Malformation of Heart.**—Much light has been thrown upon the subject of malformation generally by considering what changes would be wrought upon organs by diseased conditions occurring at an early period of fœtal life, when the parts are yet plastic, and ready to yield to any new influence; and how, also, other structures in connection with them would of necessity be modified to stand in relation with the altered parts. Such considerations have afforded many explanations regarding malformation of the heart. In speaking of diseases of the fœtus, it must be remembered we do not use the term in so general a sense as to imply a mere deviation from the normal or healthy standard of development, but we mean something much more definite; that is, that the fœtus becomes the subject of a disease similar to that which occurs in the adult, and that in consequence various adhesions or obstructions may result, which, from the parts being still plastic, and undergoing great changes during growth, produce those alterations in form that we call malformations. It is true that there are arrests of development and want of union of the two symmetrical halves of the body, and the same occurs in the heart; but still a large number of malformations appear to be due to the cause we name. That diseases do occur in the fœtus we can easily prove by examining still-born children, and especially those of syphilitic mothers, when we have often found acute inflammation in the abdomen, peritoneal adhesions, pneumonia, &c. An acute endocarditis is thus supposed to occur in the same way as in the adult. Even if the proof of this should be wanting, which we think it is not, the theory forms a good hypothesis to explain the various appearances met with in malformed hearts, and on which we can give a general interpretation of the different kinds of malformation seen on the shelves of our Museum; for otherwise it would be impossible to describe each separately, seeing that we have more than thirty specimens, and scarcely two alike. We shall, therefore, only speak of the most common, and describe the mode in which they appear to be formed.

And first it will be best to see clearly what they are not. A general impression for a long time prevailed that an open foramen ovale or ductus arteriosus is a common kind of malformation; this belief was founded upon the fact that such conditions are met with, but it was too easily assumed that an imperfectly developed adult heart would very probably show this simple kind of malformation most commonly. Such opinion we still find prevailing among students, but they must be warned against it. The most common kind of malformation, and that of which we have seen the greatest number of instances, is imperfection of the ventricular septum; such hearts often show one vessel coming off from the two cavities, the lungs being supplied by some irregular means; and with these irregularities an open foramen ovale or open Botalli's duct may coexist. Now these and similar changes may be accounted for by supposing that in early fœtal life, say about the sixth or eighth week, an endocarditis had occurred which, as we know from observation, is more liable to attack the right than the left side of the heart. Thus the pulmonary valves would become affected by an inflammatory change; adhesions or other changes then would occur among these valves, causing the partial closure of the artery. After this we can infer with some certainty what the subsequent result would be. At the time in question there is no perfect septum between the ventricles, and the blood of the right freely communicates with that in the left cavity; therefore, if any impediment should occur at the outlet of either cavity, the stream is diverted through the septum to the other side, and so the closure of the septum is prevented at the proper period of its occurrence; and thus the deficiency occurs in the wall which is seen in the specimens, where the pulmonary artery is contracted. Again, in consequence of both ventricles supplying one vessel, the aorta, the latter becomes placed over both of them, which are then of equal size, and the heart rounded in shape. If the obstruction be thus compensated for by an outlet into another channel, there may be no great stress on the auricle behind, and the foramen ovale may close as usual; but very often it remains patent, in order to relieve this chamber of the heart. And now as regards the supply to the lungs; in consequence of closure of the pulmonary artery the ductus arteriosus remains open, and thus the lungs are supplied from the aorta. The blood is necessarily mixed, and hence one cause of cyanosis and early death in such cases; although very often the duct is brought low down over the right ventricle, so that it gathers most of its blood from this side. We see, then, how an obstructed pulmonary artery necessitates all these further changes; a passage of blood into the left ventricle, and then a way out again into the lungs by the fœtal ductus arteriosus remaining patent. All these different alterations in the heart would so clearly arise from an obstructed pulmonary artery that we think there can be little doubt that here lies the *fons et origo mali.* In the three or four cases of malformation of the heart in persons who have grown to adult age, the peculiarities have been of the kind we mention.

In some cases the obstruction of the pulmonary artery is not complete, and the various conditions mentioned may be less marked, as in cases where the valves unite to form a funnel-shaped membrane projecting into the vessel, while the heart is otherwise well formed. We have several specimens of this kind, in some of which the persons had lived beyond middle age, though most died young of phthisis. In some instances the pulmonary artery is united to the aorta, and both come off from a common ventricle; in other cases, the vessels are transposed. In some remarkable specimens, although the pulmonary artery is closed or wasted away, the ductus arteriosus is also closed, but the lungs have been supplied by immensely dilated bronchial arteries. In some other cases the aorta is obstructed, and the pulmonary artery carries the blood from both sides of the heart, the descending aorta being attached to the pulmonary. This condition is seen in a heart lately found in the dissecting-room. We cannot mention all the other varieties met with, but we frequently find that the septum ventriculorum is imperfect at its upper part without any other alteration; when this is the case, the question often is asked whether it has arisen from disease, *i. e.* from endocarditis and softening of the muscular structure, or malformation; but these suppositions are not to be taken as mutually exclusive, for assuming that malformations arise from disease, it is possible to conceive that such disease occurred in the fœtal state, this condition being the remnant of it. We stated just now that the old notion that malformed hearts were due to want of closure of the openings natural to the fœtal state was erroneous, and we now see how such open channels necessarily follow from previous obstruction, and constitute conservative results rather than morbid states; still, however, we occasionally meet with open ductus arteriosus without any of those obvious causes to which we have alluded, though it is possible that these causes may have existed for a time, and have afterwards disappeared, the effect alone remaining. We should mention, however, that a simple patency of the foramen ovale is not uncommon, being met with in about one in every thirty cases. Indeed, a partial opening extends to a much later period of life than was formerly supposed; it has been found that the bodies of children show a larger proportion of examples of this condition as the children are younger, so that it is probable that the completion of the closure is a process that occurs gradually for years after birth; the opening, however, is valvular and incapable of allowing the passage of blood. Sometimes the membrane closing the opening bulges out; and occasionally it is cribriform.

We have several malformations of the heart which depend on deficient development, found only in the fœtus or children who survive their birth but a short time. Thus sometimes a heart is met with having only one auricle and ventricle. Such a bilocular heart, in spite of its low development, may show sufficiently perfect formation of the great vessels. Such cases do not grow to active life, but may continue to exist as sucking infants for a few months. Cases of trilocular heart, with two auricles and one ventricle, may reach adult life. Occasionally, too, we meet with instances where the right ventricle is divided by a septum, thus multiplying the natural number of cavities; lower degrees of this division are not very uncommon. This is due to the two portions —the conus arteriosus and the sinus of the right ventricle—remaining distinct. In one case the heart protruded from the chest, owing to the absence of part of its wall, a state known as *ecstrophia cordis.*

**Atrophy of the Heart.**—This may arise from various causes. Some authors speak of concentric, simple, and eccentric atrophy of the heart. When the heart is wasted, if its cavity be small, this state would be called concentric atrophy; if the cavity be normal in size, simple atrophy; and if the cavity be large, eccentric atrophy. The symptoms of these states are given by some; but we have not been able to find any evidence that these conditions are of the least importance, and have never known such "atrophies" to have any share in the cause of death. It is true that the small heart of an emaciated person may be found contracted or dilated; but this depends entirely on the mode of death. Simple wasting is found in emaciated persons as part of the general marasmus; but is especially seen in those who have died of cancer, when the heart is found of very small size. Thus, in a recent case of cancer of the stomach, the heart weighed only four ounces. We do not think there is any obscure connection of this atrophy with cancer; but the wasting of the heart is proportioned to the wasting of other parts; whereas, in most other wasting diseases, especially in phthisis, the implication of the lung induces a resistance to the flow of blood, and a consequent comparative hypertrophy, so that the heart generally has its normal weight. If any part of the heart receive a deficient supply of blood, it wastes; and thus, when the mitral orifice is small, the left ventricle is small, unless some other cause of enlargement is present. Pigmental degeneration as a cause of atrophy we shall refer to in a later paragraph.

Sometimes the organ wastes if there is much external fat. Such hearts do not look small. The subjects of the change die suddenly, and not slowly by dropsy. In one very interesting specimen the heart was very small and thin-walled. It weighed but five and a half ounces. The thin walls are chiefly composed of fat. It came from a woman who died of gradual cardiac dropsy, such as results from common obstructive heart disease. We look on this case as a very rare example of fatty atrophy of the heart. It has been said that fatty heart leads to dropsy and the other conditions of cardiac obstruction; but that statement does not accord with our experience. Indeed, this is the only example of dropsy from fatty disease of the heart that we have ever seen.

**Hypertrophy of the Heart.**—Simple hypertrophy of the heart is not often found post mortem, except in one disease, in which it is frequent enough. This disease is granular kidney. The heart, in granular kidney cases, is nearly always enlarged; it may reach eighteen to twenty-four ounces, or even more, but twenty ounces would be a considerable hypertrophy. In such cases there may be no dilatation, but a simple hypertrophy. We must not say with Rindfleisch that hypertrophy is invariably associated with dilatation; a twenty-ounce heart may have no cavity at all after death. But it is important to notice that all examples of hypertrophy due to valvular or other primary cardiac disease are usually associated with dilatation. This constitutes a distinction of great importance, because dilatation diminishes the efficacy of the hypertrophy, increasing as it does the surface which the stronger muscle has to operate upon in contracting. Niemeyer, in looking for changes in the muscle to explain dropsy in simple dilatation, appears to forget this fact. From many careful observations we are convinced that the symptoms ascribed to hypertrophy are due to dilated hypertrophy, and are severe in proportion to the dilatation. Patients with granular kidney and simple hypertrophy are not aware of any change in the heart. They are always adult, and often their costal cartilages are so stiff, and their lungs so expanded, that the small increase of bulk which a simple hypertrophy causes is not to be discovered. As soon as dilatation sets in then begin the symptoms, and the trouble is in proportion to the dilatation. Simple hypertrophy does not cause symptoms, and rarely exists as a primary cardiac disease.

In Bright's disease, so far as we have observed, hypertrophy is limited to the left ventricle, or nearly so limited. The right appears to share a little in the enlargement, but this is probably due to the fact that many of the fibres extend across both ventricles and no doubt must undergo hypertrophy in their whole length, so as to thicken both ventricles. The convexity of the enlarged left ventricle often encroaches on the right. The hypertrophy in Bright's disease is probably due to resistance to the passage of blood through the vessels of the general system, and the excess of such resistance may at last lead to dilatation of the heart. The renal symptoms are then complicated with and are soon outweighed by the cardiac. Aneurysm of the aorta, not involving the semilunar valves, does not seem to produce any change in the size of the heart.

**Dilatation with Hypertrophy.**—It is usual to arrange the various forms and varieties of hypertrophy and dilatation as follows: *hypertrophy* is spoken of as *simple* when there is a mere addition of muscle to the exterior of the walls; as *concentric* when it is at the expense of the interior; and as *eccentric* when the cavity is enlarged. As regards *dilatation* there is that which is called *simple dilatation*, *dilatation with hypertrophy* of the walls, and *dilatation with thinning* of the walls.

The six different forms of enlargement just enumerated can easily be reduced to four, for it will be seen that two are identical—the eccentric hypertrophy and dilatation with hypertrophy; and it is more than doubtful whether there is such a condition as concentric hypertrophy, so that this also may be struck off the list. The cases of supposed *concentric hypertrophy* have generally occurred in persons who have died of granular kidney or of thickened right heart in cases of malformation; here the heart is found empty, and the ventricle firmly contracted on itself; the cavity being thus obliterated, an appearance of hypertrophy is given to it, which would suggest the increase of its thickness by growth of fibres inwards as the name infers; but if such a heart be kept for some time it will relax, and a cavity be formed in it as usual. Thus, so far as the hypertrophy is concentric, it merely shows a mode of death.

As to the four other conditions—we have already spoken of simple hypertrophy—those that remain are simple dilatation, dilatation with thickening, and dilatation with thinning. Of acute dilatation of the heart we shall presently speak. In chronic dilatation hypertrophy almost always exists, as proved by the increase of weight which dilated hearts always show. There are, therefore, only two conditions of which it is important to speak, viz. dilatation with thickening, and dilatation with thinning, of the heart wall.

When the left ventricle is much stretched, the term aneurysm has been used; but, though we have avoided it, yet in some instances we can scarcely adopt any other name, as in an example in our Museum, where the left ventricle is immensely distended, and the walls thinned into a membrane scarcely thicker than a bladder. The analogy with aneurysm is generally maintained in these cases by the occurrence of ante-mortem coagulation of blood in the dilated cavity; but the state differs from aneurysm in this essential point, that it does not produce pressure as a tumour.

In hypertrophy without dilatation, or with little dilatation, the muscular walls may be uniformly thickened, and the trabeculæ or columns within participate; this is remarkably seen on the right side, where these muscular bands are often of great thickness, especially those columns that connect the free wall of the right heart with the septum, called by Mr. Wilkinson King "moderator bands," from their function in restraining excessive dilatation. In the left ventricle the thickness is generally of natural proportions when the hypertrophy is without much dilatation; but if combined with dilatation, the walls are thicker at the base, and become gradually thinner towards the apex. In these cases the mitral fleshy columns are not correspondingly enlarged, but have a tendency to become narrower or pointed towards their upper extremity, and to change to fibrous tissue at their summit. It appears that this fibrous change of the musculi papillares has a conservative tendency, for if the cavity of the ventricle be dilated, the tendinous cords of the mitral cannot reach across it unless they are elongated in proportion to the increase of the heart's diameter, so that the valve-flaps in such dilatation are drawn down in the systole instead of moving across the mitral orifice. Such stretching down of the mitral columns and cords is a common cause of mitral regurgitation in dilated hearts, notwithstanding that the valves and cords may enlarge and elongate to some extent to suit the new conditions. Indeed, one may say that all greatly dilated hearts always allow mitral regurgitation in consequence of insufficiency in the length of the mitral tendons. It is plain, therefore, that the change of the column to fibrous tissue, and consequent persistence of its length during systole, has a conservative tendency. But when the overstretched mitral cords break, then the regurgitation becomes much more severe and irrecoverable, the broken cords get covered with fibrin, and the friction of this extends the mischief. Thus arise most of the cases in which mitral disease complicates dilated hypertrophy of the left ventricle.

The altered conditions, therefore, of the cavities of the heart found in chronic disease are three: a simple thickening of the walls, a dilatation with thickening, and a dilatation with thinning. These conditions are found in all parts of the heart, but are especially noticeable in the left ventricle. The right ventricle being usually simply thickened or dilated with thickening; the right auricle generally dilated with slight thickening; while the left auricle is considerably hypertrophied with its dilatation.

The three most usual changes in the form of the heart are as follows: Enlargement of the right side from *pulmonary obstruction*, as in emphysema (we distinguish this from simple wasting of the lungs in which the heart is small) or cirrhosis of the lungs, in either of which diseases the right heart widens and enlarges from inability to empty itself, while the left remains small or even shrinks, always shortening a good deal, so that the right heart coming in front of and below it forms the apex of the whole heart. Thus, the organ is broad; or, in fact, has more or less a rounded form instead of being conical. It is important to recognise this, for it tells a tale of long-standing pulmonary obstruction. In emphysema, through the large lungs depressing it, the heart lies more in the middle line of the chest, and lower down than when the left heart is enlarged. In cirrhosis of the lung it is displaced towards the diseased lung.

In cases of contraction of the *mitral orifice* the figure of the heart is at first sight much the same as that just described, for the obstruction soon is felt through the gorged lung in the right side of the heart, but the left auricle first becomes overcharged, consequently distended, and its walls hypertrophied, so as to reach often a very great size, while its walls become remarkably thick and tough, and uniformly so throughout. Thus, the heart in contracted mitral orifice is rounded from enlargement of right side, and with this there is an enlarged left auricle. In simple obstruction of the auriculo-ventricular orifice, the left ventricle is found smaller than natural, but often some other diseased condition, casually associated, gives rise to its enlargement.

When, however, the *stenosis occurs in childhood*, the amount of blood passing through the heart is diminished in quantity, and the whole organ is small in proportion; the left ventricle with the aorta is small, the left auricle not enlarged or but slightly so, and the tricuspid is also narrowed like the mitral. The lungs are diminished in size together with the trachea, and the whole chest is diminutive. The mitral is, in fact, the measure of the amount of blood circulating through the system, and this has been curtailed. Such cases as this are occasionally met with on the post-mortem table, where there have been no symptoms whatever of heart disease. These facts were detailed by Dr. G. H. Barlow in his Goulstonian Lectures, but with a different interpretation. He believed the narrow or malformed chest was the first factor in the series of events, and the heart with its orifices became reduced in size owing to the lessened circulation through the lungs; we cannot, however, agree with this view of the case.

In *aortic disease* we have immensely enlarged or *bovine* hearts, as they are sometimes called. Owing to the obstruction at the mouth of the aortic orifice, and the subsequent reflux of the blood, the left ventricle becomes often immensely distended, and its walls thickened. In young subjects the large heart may then obviously bulge out the left chest, the intercostal spaces over the heart being widened. The left ventricle elongates, so that the heart is longer, and it extends further downwards and to the left; we do not find its axis more transverse through gravitation as some say; the connection with the trachea prevents this. If there be much aortic regurgitation, and the muscle be poor in texture, the ventricle is often much thinner at its apex than at its base. All the other parts participate in their turn in the consequences of this impediment to the flow of blood through the left ventricle; the left auricle becomes unable to empty itself, and the effect of the obstruction extends round to the right side, as just now mentioned, so that the whole heart becomes enlarged.

Besides these examples of enlargement from obvious causes, we meet with cases of *simple enlargement* without any valvular disease, and these constitute, generally, the largest hearts found in Museums of which we have examples. In some of these the orifices are increased in size, but this may be a consequence of the ventricular distension. Thus the left auriculo-ventricular orifice is enlarged, and probably allows the blood to regurgitate, for the ventricle is often greatly distended at its posterior part behind the valve. The frequent association of this simple form of enlargement with Bright's disease and rigid arteries suggests whether it be only a further stage of the ventricular hypertrophy already mentioned, and that, owing to the obstruction being greater than the hypertrophy is able to overcome, the hypertrophied left ventricle cannot empty itself, and dilates, while its gorged condition obstructs the lung and right heart until the usual hypertrophic effects are brought about in these. Dr. Gairdner thinks such enlargement may be due in some cases to a contraction of the lung from old pleurisy, so that the lung cannot fill the chest, and hence the heart expands through the disproportionate suction exerted on it in the act of inspiration.

We have met not infrequently with cases which have convinced us that dilated hypertrophy of the *right heart and pulmonary arteries* must be distinguished as an important and idiopathic disease. Sometimes the right heart is of enormous size, and the pulmonary arteries dilated, so that the third division of the branching produces arteries as large as or larger than the normal main trunk, while the main trunk itself is two inches in diameter. The small arteries throughout the lung stick out on section like crow-quills. We have in such cases searched vainly for any condition of lung to explain the dilatation of the heart, and are obliged to conclude that it is idiopathic.

In a case given in vol. iii, Series 3, of the 'Guy's Hosp. Rep.' of a woman who died with all the symptoms of cardiac disease, the right ventricle was twice the size of the left, and immediately above the sigmoid valves the pulmonary artery dilated to five inches in circumference and continued this size into both lungs, each branch being twice the calibre of the aorta. Four fingers could easily be passed into the vessel, which was much affected with atheroma. A lesser degree of dilatation of the pulmonary artery is of not infrequent occurrence.

*Acute dilatation of ventricle and heart.*—Dr. Barlow was in the habit of enforcing the doctrine just mentioned that the same causes which give rise to hypertrophy in Bright's disease may also lead to dilatation. His views may be found in a paper published in the 'Reports,'* in which he says that dilatation may be produced not only by ordinary valvular obstruction but from changes in the quantity or physical properties of the blood which pass through the orifices and channels, and that circumstances may prevent the occurrence of the more usual hypertrophy. Where, he says, there is considerable deficiency in the activity of the nutritive function, the hypertrophy which is the natural and within certain limits the compensating result of obstruction cannot ensue, and then the cavity yields and becomes enlarged, or, as we term it, dilated.*

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* 'Guy's Hosp. Reports,' Series 3, vol. v, p. 348.

Subsequently Dr. Goodhart, by independent observations in the post-mortem room, came to the conclusion that the sudden deaths which occur not infrequently in scarlatinal dropsy are due to an acute dilatation of the ventricle. He several times found in children who had died rapidly a widely dilated ventricle full of blood. He believed the same cause to be in operation as produces the hypertrophy in Bright's disease, that is an obstructed peripheral circulation owing to the morbid state of the blood, and that the scarlatinal influence had weakened the heart so as to cause it to give way under the abnormal strain. His conclusion was that an acute nephritis, especially of the scarlatinal kind, would give rise to the dilatation, owing to the peripheral obstruction combined with the degenerative changes in the muscle.

It may be as well to mention here, in connection with hypertrophy, the changes which take place in the muscle. The question has long been asked, do the muscular fibrillæ grow larger in hypertrophy, or are new ones formed ? There are some who think that the fibres actually grow in size, but of this there is some doubt, although there can be none as to their increase in number. We have often examined hypertrophied hearts, and have found great difficulty in making out the points in question; but we have seen nucleated fibres which probably were the germs of new tissues. The hypertrophy of the right ventricle differs remarkably from the left : if we feel the two ventricles of a hypertrophied heart we perceive that the left can be readily torn through by the finger, while we cannot do this to the right without considerable difficulty. Why this tough leathery consistence should occur on the right side and not on the left is not altogether clear ; whether the original texture on the two sides is in any way different, or whether there is any condition of the mechanism of the right ventricle which may account for it, we have not yet determined. It has been stated, but without any sufficient foundation, that there is found an inflammation of the perimysium and production of connective tissue. It is important to recognise this change of consistence in the right heart, for thus we are able to ascertain the hypertrophy when the thickening is scarcely visible.

**Obesity of the Heart.**—This is met with mostly in persons above middle age who have grown fat, and then it forms part of a general fatness, bringing with it, however, special dangers. Sometimes it is found in persons who are old and wasted, and the same disposition is seen in those who have been intemperate, especially those accustomed to an excessive use of malt liquor. In such we may find the front of the heart wholly covered with fat, so that only a small portion of the muscle of the right ventricle can be seen; and, on cutting into the cavity, we find that the adipose tissue has penetrated the muscle and streaked it throughout, or by its encroachments it may have actually destroyed it, especially in a part of the wall of the right ventricle. This is generally at the apex, but sometimes at the base also. We then find that the whole thickness of this part of the right ventricle is made up of fat, and we can see, by looking at the part closely, where the layers of muscle run into and lose themselves in the fat. Under these circumstances the muscular fibres generally simply waste away by the pressure of the fat. Examining sections by low microscopic powers we see the fat growing in the course of the vessels; we can see this also plainly on the surface with the unaided eye.

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* 'Guy's Hosp. Reports,' Series 3, vol. xxiv, p. 153.

The whole right heart may be covered with fat. On one occasion it reached the thickness of half an inch, the thinnest part being one third of an inch thick over the middle of the right ventricle; but this quantity of fat is very rare. We have never seen the left ventricle covered all over with fat. Obesity is sometimes associated with great atrophy of the heart. Thus, a heart thickly covered with fat may weigh only seven ounces.

**True or Saccular Aneurysm of the Heart** is that form of disease in which there is a sacculated pouch from one of its cavities. The most marked cases are those where the pouch is circumscribed and communicates by a small opening with the larger chamber within; in these instances there has generally been no history of acute inflammation of the heart, and therefore there is no proof that they result from such a process; they are found often towards the apex of the organ, while the acute aneurysms we shall speak of under acute endocarditis arise mostly in the neighbourhood of the valves. Those in question seem rather due to a chronic process, and are formed like aneurysms in the blood-vessels, or still more like the saccular pouches of an hypertrophied bladder, which are called "herniæ" of its mucous membrane. The heart, like the bladder, shows an uneven surface within, areolated with columnæ carneæ. The spaces between these are weaker parts of the wall. Such a weak space may yield when the heart is a little dilated, forming a pouch at first in the wall of the heart, which as it increases projects on its outer surface, yet, even if large, it still communicates with the ventricle only by a small hole representing the little area between the columns which originally yielded. So far the formation of the saccular aneurysm of the heart is exactly like the sacculation of the bladder wall, and a degree of such sacculation is not infrequent in dilatation, favouring ante-mortem coagulation. But a fibrous degeneration appears early in the wall of any large pouch. We believe that in the kind of cases we are now speaking of, where the sac communicates by a small hole with the heart, the process begins like sacculation of the bladder, and the fibrous and calcareous degeneration follows afterwards. Dr. Fagge, however, collected a large number of cases, and finding them always associated with the fibroid changes, believed that their origin was contrariwise, namely, that that a fibroid deposit took place in the walls of the heart, and that this softened and gave way, causing a vacuole or aneurysm. In one remarkable case there were several pouches or multiple aneurysms. These pouches are mostly found at the apex of the left ventricle, though sometimes in the auricle, especially the left; the ventricle is itself enlarged and atrophied, with the thin-walled sac coming off from it. One of our best specimens came from a patient who died of phthisis. It was found accidentally, and had undergone changes which had resulted in a cure. It was situated at the apex of the heart, and was firmly united to the pericardial sac. It was about the size of a pigeon's egg, of an oval shape, its walls bony and very hard, and communicating with the ventricle by a small opening which would only admit a quill. Its interior was occupied by a soft translucent matter, which appeared to be fibrous, and had undergone considerable degeneration. We met recently with another example exactly like this in the possession of Mr. Townsend, which is now in our Museum; here the aneurysm had undergone complete cure.

Dr. Southey had a case where an aneurysm at the apex grew to the size of the heart itself, and immensely distended the pericardium.

We have two specimens showing aneurysm at the undefended space of the septum.

**Injury and Spontaneous Rupture of the Heart.**—Although two distinct conditions are here referred to, we place them together in order that they may be contrasted, as their recognition is of great practical importance. The one is rupture of the heart from direct violence, and the other that which arises spontaneously; the correct knowledge of the characters of spontaneous rupture may be of the greatest consequence in a trial for manslaughter. A person, for example, is seen to fall in the street amongst a number of vehicles; he is picked up bruised and dead; a rupture of the heart is found, and the question is asked whether this is due to direct violence or from emotion during fright. Spontaneous rupture of the heart, in nearly every case, arises from a softening of the tissue, and may be considered, therefore, as indicating generally a diseased organ. But in a specimen taken from a brewer's drayman who died suddenly while lifting a beer barrel, and in whose left ventricle there is at the back near the apex a rent two and a half inches long, we cannot discover any important change in the muscular substance of the spirit-kept preparation. Spontaneous rupture occurs, for the most part, in the left ventricle. The other parts, especially the right ventricle and auricle, are less frequently affected; whereas the part of the heart most liable to injury by violence is the right ventricle, or front part of the heart, as might be expected. Other parts may, however, suffer. Thus, we had a case of traumatic rupture of the left auricle just below and internal to the apex from a fall in which the sternum was not broken. Rupture from disease occurs mostly in persons advanced in age, in whom a fatty degeneration has taken place; and the laceration occurs generally in the left ventricle, towards its apex, by a slit more or less transverse, running in the course of the fibres, and sometimes an inch or two long; in some exceptional cases the rent has reached the septum. Sometimes the laceration, from either cause, is merely on the exterior of the heart, and has not penetrated the interior, as seen in a specimen, where two or three superficial rents are seen, and death occurred from hæmorrhage into the pericardium. Sometimes, as in another specimen, the laceration is limited to the inner layers.

The differences we have just enumerated enable us to form a nearly certain judgment in cases of rupture of the heart as to its spontaneous or traumatic origin. Most examples of traumatic rupture are parts of some very serious injury which is otherwise manifest enough, for the healthy heart is not ruptured except by extreme violence. The most stable grounds of conclusion in spontaneous rupture are drawn from the heart itself, while the conclusion is rather drawn from the coexistent conditions in traumatic rupture, for the rent through injury may happen in almost any part of the organ, although less likely to occur in the left ventricle, *i. e.* the part most liable to spontaneous rupture. In speaking of the degenerations of the heart we shall show some reasons why the left heart suffers most in all spontaneous disease. We have met with some cases which proved that spontaneous rupture of the heart had occurred at two stages with at least several weeks' interval.

**Punctured and Gunshot Wounds.**—These are by no means invariably at once fatal, patients living sometimes for weeks after such injuries, while slight wounds may probably be recovered from. The injury, if not fatal at once, produces death by the subsequent inflammatory results. In a specimen, which was given us by Mr. Callaway, the heart is traversed by two bullets; but the man spoke after the receipt of the wounds. There was a patient in the hospital, who was stabbed through the pericardium, although the heart appeared untouched: the man lived two days. A curious case was recently shown at the Clinical Society, where a needle, which had been driven into the chest, moved with the action of the heart so as to convince the observers that the needle was in the heart itself.

In one case a needle passed through the bronchus into the heart, and in another through the œsophagus into the same organ. A case was reported from Birmingham where a man lived four years after a stab in the heart.

**Effusion of Blood** is sometimes met with in the substance of the heart in cases of purpura, or other diseases where the blood is affected. Also beneath the endocardium, on the interior, such purpuric patches may be seen; and this has long been noticed as occurring in cases of poisoning by arsenic and mercury, and especially by phosphorus. In cases we have examined in this hospital after deaths caused by all these kinds of poisoning patches of ecchymosis were found in the right auricle and left ventricle. It may also be due to infarction, the vessels being blocked in connection with embolism elsewhere, and may also occur from spontaneous thrombosis in the coronary vessels.

## CHANGES IN THE MUSCULAR FIBRES

**Passive Changes.**—*Softening.*—The heart is found soft when its *rigor mortis* has passed off, as students' experience in the dissecting-room teaches them, where the heart is generally almost pulpy, by the time it is reached in the usual course of dissection. At the same period the spleen is very pulpy, and the vessels contain air bubbles, while their interior is deep red or purple from imbibition of serum stained red through solution of the corpuscles in it. These are phenomena that accompany decomposition, as we all well know. Now, in some cases the softness of the heart is very remarkable when there is no sign of decomposition about the body generally. When this is the case we may find the spleen more or less pulpy, and the vessels deep red within, and containing air just as in advancing decomposition. The course of the vessels under the skin is then marked by purple lines, and the trachea and bronchi are stained intensely red. We have known this change supervene in five hours after death, and that in winter time, when the weather was cold, and when no signs of decomposition appeared in other bodies kept for several days. By considering the kinds of cases in which this early decomposition of the blood, and softness of the heart and spleen appear, we shall find that it is more common in fevers, in persons who have died obscurely in two or three days after great surgical operations, or in persons who have died by coma in jaundice; that is, in short, where there are other reasons to believe that death was caused by changes in the blood. Speaking generally, we should say that marked softness of the heart is an index of changes in the blood, and goes with pulpy spleen, red endarterium, and at last air bubbles in the vessels.

Such pulpy softness of heart is of great importance among the signs of blood changes; so that when it supervenes very early after death, and the rest of the body is free from decomposition, there is great probability that the unusual condition of the blood thus declaring itself was in progress during life—we would not say to the extent of forming air within the vessels, but this may happen. We have had occasional evidence that emphysema from decomposition, without any gangrene, may be found commencing during life, and the same gaseous exhalation may continue after death. In these cases the softness of heart, &c., may come on exceedingly quickly, being complete in a few hours.

But we have no knowledge of the state of softening of the heart to which some authors on the heart devote a chapter, ascribing special symptoms to it. Softness of the heart is important, but its importance is indirect as an evidence of febrile blood changes—blood solution.

*Pigmentary degeneration.*—The heart is sometimes brown and friable, having less consistence than is natural. The fibres show yellowish-brown pigment grains, which at first are seen about the nuclei of the fibres, and afterwards in streaks within the fibres. This condition is, according to our experience, very frequent, and is not a sign of any primary or special disease of the heart, but belongs to senile states and general emaciation from any cause. We would mention that this brown change is not found in the voluntary muscles.

*Fatty degeneration.*—By this we do not mean obesity of the heart already spoken of, but a change in its muscular fibrils, such that their interior becomes loaded with fat, which obscures their striation until perhaps the fibres look like columnar aggregations of little bright round granules. Treated with osmic acid they become dark brown or black. This change may exist without any increase of external fat, and may occur in a person otherwise thin; it is also found often accompanying valvular disease of the heart. It may present either of two kinds of appearances—one where the whole muscle is affected; and the other where the inner surface of the heart is marked in a peculiar manner. In the latter condition the rest of the muscle is often of a good colour, and the external part healthy, but within the ventricle the heart is seen to have a peculiar streaked appearance; on looking carefully at it a number of white transverse zigzag lines are seen running parallel to one another, producing a feathery appearance, or rather they look not unlike the markings on a tabby cat. They are seen especially, and in the first instance, on the mitral columns of the left ventricle; afterwards they appear on other parts of the cavity, and in extreme cases may be seen in the right ventricle. We have never seen this change in the auricle. It only penetrates a slight depth in the substance of the heart. The question arises how long is such a process in formation, and what are its exact pathological indications? It is found in connection with other disease of the heart, and is especially well developed and obvious in those cases of simple anæmia which have been called idiopathic for want of other explanation. This condition cannot be shown in our Museum preparations, for it altogether disappears after the organ has been kept a short time in spirit; but we shall, no doubt, soon be able to see it in a recent heart.

In the more general form which affects the whole thickness of the heart the muscle is found yellow-looking, flabby, and easily lacerable. This is the form of fatty heart which has long been known to be often associated with disease of the coronary arteries as a cause of *angina* *pectoris.* Although this is the most usual change, it seems scarcely sufficient to account for the symptoms. The paroxysm has been thought to be due to a spasm of the coronary arteries from change in the cardiac ganglia or nerves, and Dr. Goodhart has lately argued that the disease is essentially a muscular cramp, a somewhat unusual, but nevertheless a well-recognised consequence of muscular fatigue.* Sometimes a limited part, usually towards the apex of the left ventricle, is much more affected by fatty degeneration than the rest; it may be found, indeed, breaking down. The branch of the coronary artery going to this part is then generally more diseased than the rest; indeed, some cases have occurred where such a localised fatty patch, with its coronary artery very badly diseased either by atheroma or embolism, has been found in a heart that was sound in other parts. Such a condition, combining power in the whole heart with weakness of a limited spot, strongly predisposes to spontaneous rupture at that weak spot; while a heart, whose ventricle is everywhere equally fatty, though less liable to burst, makes up for this by its tendency to stop suddenly. A small yellow soft spot may sometimes be found in connection with infarction of the vessels.

Some surprise was created by the observation of Dr. Hermann Weber, who stated that on chemical examination hearts in this condition present no increase in the quantity of fat present. The amount of fat is certainly very small as compared with the amount in fatty degeneration of the liver and other organs. In an analysis kindly made for us by Dr. Stevenson five per cent. of the recent muscle was found to be composed of fat, which is about twice the quantity present in heart-muscle that is microscopically healthy. This result agrees with those obtained in an elaborate series of researches by Krylow, which showed that although the amount of fat is always small in the change in question (four per cent. of the fresh, or twenty per cent. of the dry muscle), yet there is relatively a decided increase. Nevertheless, the amount of increase is positively so slight as to make it probable that the granular appearance of the muscular fibres is due rather to a rearrangement of their elements than to a large addition of fat, as was supposed before Dr. Weber's researches were published.

**Active Changes.**—*Inflammation; diffused myocarditis of low degree.*—It is highly probable that in those very common cases of inflammation of the exterior and interior of the heart accompanying rheumatic fever the muscular structure itself in often involved. We think so for many reasons; firstly, in hearts with pericarditis and endocarditis we have met with proof, many years afterwards, that the muscle had been affected in the fact of this structure being occupied by an adventitious fibrous tissue; secondly, the enlargements which occur after such inflammations, with only slight or with no valvular disease, cannot be accounted for except by supposing some altered condition of the muscle; thirdly, in fatal pericarditis, if the adjoining muscular tissue be examined, the fibrillæ will be found fatty; fourthly, we have clinically observed the heart to enlarge and its sounds and beat to become almost imperceptible during the course of rheumatic fever in some cases; and, lastly, it is only on the supposition of some alteration in the heart that we can account for certain cases of sudden death which occasionally occur in the course of that disease. It is not remarkable that in cases of speedy death no very marked changes beyond softening should be observed in the muscle; and we think, nevertheless, that there is such an affection as an *acute general inflammation* of the muscle. A further stage would show a loss of striation and granular fibrillæ.

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* 'Guy's Hosp. Reports,' Series 3, vol. xxix, p. 335.

*Ulcerative myocarditis.*—This is sometimes met with in connection with endocarditis, especially when it attacks a valve; and, owing to the changes which usually follow in the substance of the heart, the name of *acute* or *false aneurysm* has been given to it. The manner of attack is generally in one of the following ways:—An endocarditis, affecting as usual the valves, reaches such intensity that the tissue of the valve ulcerates, and the ulcer spreads to the root of the valve and invades the muscle; or a great mass of vegetation attached to a valve by its direct pressure cuts into the muscle, forming an ulcer; or, what is more frequent, a mass of fibrinous clot attached to the moveable part of a valve, and swinging in the blood current, comes repeatedly into contact with the heart's wall, and so by friction starts an ulcer; the muscular tissue becomes involved, and the ulceration spreads in the muscle more or less rapidly, so that an excavation is formed generally near the root of a valve.

However formed, this excavation of course communicates with the ventricle, and its contents mix with the blood, so producing very terrible consequences. For, carried in the stream, these products, if the ulcer is intense, set up the so-called infarctions leading to destruction of portions of the organs; or by acutely inflaming and destroying the wall of a considerable artery cause acute aneurysm of it.

When examining the ulcer microscopically we see the muscular tissue of the heart sometimes only exposing a ragged surface with little infiltration of the substance; but sometimes the floor of the ulcer is charged with pus to a depth extending through the whole thickness of the left ventricle. We found in one such case portions of muscular tissue still plainly recognisable, and adhering to the surface of the ulcer so lightly that a few more beats of the heart must have thrown some of its muscle into the brain or elsewhere.

When such ulcers are present in the heart we always find larger masses of friable, half degenerated, fibrinous clots, perhaps partly calcified, forming masses on the valves near the ulcer, and we nearly always find that these masses of fibrin are so placed as to give rise to the ulcer by friction.

As the ulcerative process proceeds outward, the visceral pericardium may be softened, and an acute pericarditis set up, as seen in a preparation from a case which occurred only a short time ago in the hospital. The patient was a young girl who had acute endocarditis; and the muscle was involved close to the outer aortic valve. The bursting of such a sac might possibly cause sudden death by hæmorrhage into the pericardium. The softening or abscess need not necessarily take its course outwards in this direction; for sometimes it proceeds between the valves, and forms a small aneurysm there; sometimes such an aneurysm may eventually burst upwards, between the ventricle and auricle; or, if the process be towards the septum, a pouch may be there formed, or an actual perforation; as in a case where the acute ulcer at the root of the aortic valves had pierced through into the *conus arteriosus* close to the pulmonary valves. Many morbid processes are very active at this "undefended spot."

Endocarditis in this acute and grave form constitutes a disease which is totally different in its history from the obstructive form of heart disease. The sufferer dies generally without dropsy, often suddenly, and the case is marked by its subfebrile course, and the occurrence of paralysis, aneurysm, or the other evidences of embolism. It mostly occurs in a chronically diseased valve.

As usual in the heart diseases of extra-uterine life the left side, *i. e.* the hardworked side, is that which suffers most; but ulcerative endocarditis is met with on the right side, especially, according to Dr. Carrington, in puerperal cases.* The disease is more apt to extend from the aortic than the mitral valve. In two cases the illness was ascribed by the sufferers to excessive drinking; but these men had been soldiers, and it is known that military exercise overstrains the heart. It has appeared that overstrain has predisposed to, and rheumatism or alcohol excited, this grave disease.

*Abscess of the heart.*—This arises under conditions quite different from those which produce ulcer. A few cases are recorded of spontaneous suppuration diffused in the tissue of the heart; but we have never met with this. Abscess of the heart is practically always pyæmic, and it is seldom met with except in cases of violent and general pyæmia. We have seen several cases, and they have mostly been in youths who had osteo-myelitis of the long bones, where the kidneys also contained purulent deposits. The immediate cause of death in these instances is often a pericarditis from the bursting of small abscesses into the pericardium. Besides the lymph covering the serous membrane, there is seen a number of small points of purulent matter within the substance of the heart itself. The character of these is peculiar, and resembles similar deposits in other parts, in their being surrounded by an area of congestion, which seems to indicate, as we shall hereafter mention, that this form of inflammation arises primarily from obstruction of the blood-vessels by emboli, due to the presence in the blood of some of the elements of pus or other infection; for in other parts of the muscle we could see, when it was quite fresh, small red patches of congestion, constituting the stage prior to the inflammation, just as usual in embolisms.

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* 'Guy's Hosp. Gazette,' New Series, vol. i, p. 239.

Sometimes these abscesses will burst into the interior of the heart, and then the blood enters and washes out the pus which may be carried in the blood stream, producing infarctions elsewhere. It is said that abscesses in the wall of the heart may wither and become calcareous; but we have never seen any proof of this.

*Subinflammatory and fibroid degenerations.*—We have just said that the results of inflammation were seen in streaks of fibrous structure running into the muscular substance. Extreme examples of this kind are always combined with indications of previous endocarditis and pericarditis, and are allied to the changes seen in the liver, lungs, &c., where the investing capsule is the subject of chronic inflammation, involving the adjacent texture, and thus, the pericardium being thickened and adherent, the neighbouring muscular walls become involved in the fibrous change. This is shown by a section of the wall of the ventricle, as in those specimens, where fibrous bands and streaks are seen pervading the tissue in all directions. Sometimes the whole thickness of the ventricle is converted into a tough fibrous structure. When these fibrous bands are seen along with obvious signs of old pericarditis or endocarditis, they are referred to the extension of these processes into the tissue beneath; but sometimes we meet with patches of fibrous substance in the tissue of the heart, when there is no evidence of former pericarditis or of endocarditis other than the sharing of the inner surface in the change at the affected spots. The interior of the ventricle is marked with patches of a pearly whiteness, owing to a fibrous change of the endocardium, and the muscular trabeculæ beneath, when cut asunder, are seen to have a mere trace of muscular fibre in their midst, some of the smaller being wholly fibrous.

Such examples are not very uncommon, and their pathology is very interesting. Is it inflammatory? We sometimes find excess of new corpuscles in the growing edge of the patches and hence might infer that they are inflammatory. In one case we met with such tissue in the heart along with endarteritis, which had led to closing up of several of the principal arteries of the body. This would lead us to infer a general inflammation of the lining of the vascular system. But often they histologically appear only as fibrous tissue, and the existence of former inflammation is an inference unsupported by history of rheumatism, &c., which in our cases has generally been wanting. Even on careful examination of more recent cases the question is difficult to solve. But lately we examined the body of a boy who had rheumatic fever ten months before his death. The heart showed the result of a peri-, endo-, and myo-carditis. The section of the ventricle showed white streaks, which on examination proved to be muscular fibres which had lost their striation and become granular and somewhat translucent. No connective tissue was present, and the question arose whether the changes should be regarded as inflammatory or simply degenerative. Towards the pericardium the white streaks were markedly fatty. As regards the more chronic cases, where the muscle was streaky, Dr. Fagge collected a considerable number, and in these a large majority gave no evidence of any previous inflammation of the serous membrane, either within or without, although the endocardium might be thickened. The morbid process had been confined to the muscle itself, and the result was the production of a fibroid tissue, but no active growth proceeding from cells was seen. He considered, therefore, that there was no proof of the previous existence of a myocarditis, and much less was there evidence of its being always associated with a pericarditis.

Dr. Charlewood Turner presented a valuable paper and drawings to the International Congress on fibroid degeneration of the heart. He showed that with the destruction of the muscle there was an active corpuscular growth and fine reticulum of fibres. There was, in fact, a fibroid thickening of the perimysium with an atrophic condition of the muscular fibres; the blood-vessels were also thickened.

More recently Dr. Stevens, of Glasgow, has published his observations on the subject. He also makes a great distinction between an interstitial myocarditis and fibroid degeneration. In the latter case he always finds disease of some branch of the coronary artery. Some recent German writers have attempted to show that an infarctus precedes the change in the muscle, and this may be of the nature of a thrombosis or embolism. It is no doubt true that these fibroid changes are frequently found to be associated with diseased and obstructed vessels, although it must not be forgotten that degeneration of the vessels is often found with changes in the muscles.

Besides these circumscribed patches of fibroid degeneration we find in the interior of greatly dilated hearts that a fibroid change invades the *columnæ carneæ*, especially in the left ventricles. These may be changed to fibrous tissue. Such a change is probably due to a stretching of them, drawing the middle part of them away from their vascular supply, which must enter at the ends. The change is always begun in the middle of the muscular columns and at the apices of the *musculi papillares*, that is, at the points farthest from the entry of their vessels.

**Syphilitic Myocarditis.**—This is not infrequently found not only in the form of fibrous scar, but also in well-characterised gumma. Its characters do not, however, then differ from syphilitic gumma of ordinary muscles.

Our Museum contains a specimen of a large gummatous mass in the septum, but every part of the heart may be affected. Where there is merely a spot composed of cell growth, or a fibroid streak, there is nothing to render it characteristic, but if, as is sometimes the case, it is not one of fibroid degeneration, but where a number of hard nodules are found in the heart, there can be little doubt of their syphilitic nature.

**Tubercle.**—This has already been spoken of, as affecting the pericardium ; it does not, as far as we know, penetrate the heart's muscle.

**Morbid Growths.**—*Cancer* usually occurs in the heart under one of two distinct conditions, that is, either as an extension from primary cancer around, or as a secondary growth. The first of these conditions is most common ; the heart is implicated in a mediastinal cancer, which grows into the pericardium along the veins ; for these are generally implicated, while the arteries resist the disease ; hence the auricles are invaded rather than the ventricles. The extent of the cancer of the heart in these cases is generally not large, but we have seen nearly the whole of both auricles much involved ; and in one case, where, however, the primary disease was in the cervical glands and we could not be sure whether the heart was directly or secondarily invaded, almost the whole heart at first sight seemed covered with cancer. Sometimes, in our experience more rarely, though the experience of others differs here, the heart is affected with secondary formation of cancer, just as other parts might be. We have seen epithelial cancer of the skin of the neck associated with a secondary nodule in the right ventricular wall. In this case the inner face of the mass had on it two large polypoid cysts containing a clear brown fluid. The walls of these cysts were partly of fibrin and partly of cancer, and the latter ran continuously into the former so as to convince us that the fibrin was being permeated by the growth. In the case of extensive cancer of the whole heart, above noticed, there were numerous ante-mortem fibrinous polypi, and microscopic sections showed cancer extending into them. We mention these cases in some detail because great doubt is thrown by good authorities on the growth of cancer in fibrin clot.

We have already noticed the occurrence of cancerous nodules in the pericardium.

The forms of malignant growth that we have found in the heart have been various. We have met *epithelioma*, *lymphoma*, *melanosis*, and *sarcoma*.

Among adventitious growths we possess a heart having two cysts on its front. They are formed of thin membrane, containing a fluid, and are traversed throughout by a number of delicate fibres. Of what nature these cysts are we cannot say. The heart came from a lunatic, who, while attempting to strike another man, fell down dead, and the cause was found to be a rupture of these cysts into the pericardium.

We have also a very remarkable specimen of growth upon the heart, an osteochondromatous tumour, like a large mass of coral, growing upon the organ, and as large as itself. Whether this is a growth secondary to similar tumours in other parts of the body to which it belonged, or whether it be the remains of an included ovum which had become attached to the heart, we cannot say.

We possess two true polypoid growths in the heart, although these are rare. We have only these two specimens, and both are growths in the left auricle springing from the septum near the fossa ovalis. In the old preparation there may be seen a polypus, semitranslucent like a mass of size, which was injected through the coronary artery, although the vascularity is not well seen now. The other heart came from a woman who was in Charity Ward some years ago under Dr. Addison for hemiplegia ; after death a growth was found in the left auricle, and nearly filling it ; it is of an oval form, and grows from the auricular septum. It consisted of nucleated fibres, and might, therefore, be called fibrous, or fibro-plastic, and when freshly cut we remember that it was distinctly vascular. Dr. Goodhart states that he has met with a similar growth in the right auricle, and that it had no connection with the patient's death.

**Hydatid.**—This occasionally occurs in the heart, though it is rare in the human subject. The cysts grow, probably, in the muscular substance, and then cause death by rupture or pressure. In a heart from a girl who died in the hospital a few years ago the hydatid cyst is seen growing in front of the heart, between the auricle and ventricle, forming a cavity between the two. It is of about the size of an orange. The bursting of the cyst caused fatal pericarditis. In another case, the rupture occurred into the right ventricle of the heart. In one of our cases the hydatid was placed at the back of the heart over the auriculo-ventricular septum, and grew to the size of a large plum ; its pressure had completely occluded the coronary sinus, so that the opposed faces of the sinus were inseparably united and sealed up. Drs. Greenhow and Cayley, to whom this specimen was referred, showed that water thrown into the distal part of the sinus found its way into the right auricle by small openings, which were enlarged venæ Thebesii. We may mention that there are cases recorded of hydatid of the liver bursting into the pericardium. In one case a hydatid cyst was found in the heart, full of purulent pultaceous material.

The *trichina spiralis*, which infests most of the muscles of the body, and all the other striped ones, is never, or extremely rarely, found in the heart. Lancereaux describes a case where several cysticerci were found in the heart in connection with the same parasitic condition of the skin which he calls *ladrerie.*

## ENDOCARDIUM AND VALVES

**Atrophy and Hypertrophy.**—The endocardium is usually considered as corresponding to the internal coat of the arteries, but their pathological relations are not so much alike as this view of their nature might suggest. Thus, if we exclude the valves from our consideration, we may say that the endocardium does not suffer that fatty degeneration which is so very common in the lining of the arteries. Indeed, the endocardium is liable to but few changes, and in estimating those that it does offer we must bear in mind that it differs naturally in thickness in the different cavities of the heart. It is much thinner in the right side of the heart; indeed, in the right ventricle it can only be recognised by the polish its presence produces. On the left side its thickness is greater, especially in the left auricle, where it is naturally opaque and whitish.

**Inflammation.**—*Distribution of endocarditis.*—It will be noticed that except obesity, which may not be disease, all the diseases of the heart occur far more frequently on its left side. This is true of the fibroid patches, syphilitic or otherwise; also of the fatty change, both that occurring in streaks and that permeating the whole thickness in the overworked hearts of aged people. Thus, we may say that the strongly acting and hard-worked ventricle suffers most from disease. We shall go on presently to show that the seats where endocarditis declares itself in the left side of the heart are almost invariably places where friction is exerted, and we shall have to point out that the rarer cases, where endocardial thickening and inflammation occur in the right heart, are always cases of unusual hypertrophy of it; where it exerts more mechanical power and, therefore, causes more wear and tear of the structures connected with it. All this evidence goes far to prove that the reason why the left heart suffers more than the right is because it is stronger and works harder, straining the mechanism of its valves, and irritating its lining membrane with the friction of its blood upon it, while the right heart escapes because its play on its contents and valves is milder and less forcible. As a further proof of this we may remember that in fœtal life, where the right heart is doing all the work, any disease almost always attacks the right side of the heart.

*General characters of endocarditis.*—We next note that when we speak of inflammation of the endocardium or endocarditis, we do not imply that any such process ever occurs all over the endocardium, as pericarditis is found all over the pericardium, or peritonitis all over the peritoneum; that is, we never find the whole endocardium even of a single cavity coated with lymph or other products of inflammation. The inflammation is always circumscribed. We may occasionally find several such patches of endocarditis, discoverable by granulations, or fibrin, on the membrane; but, except in the rarest cases, these patches are within reach of a fibrinous clot on a valve, which no doubt struck the affected part in the action of the heart. When considering the possibilities of such contact in any specimen, we must bear well in mind how the heart closes its cavity in contracting, and thus brings together parts that in the dilated and dead hearts are remote from each other. The study of a great number of cases has led us to conclude that such friction, with fibrin clots, together with mechanical strain, make the principal if not the sole *direct* causes of endocarditis; rheumatism and other general states creating only a vulnerability of the fibrous structures, so that they cannot resist the irritation of the friction.

*Comparison of endocarditis with other inflammations.*—When inflammation was regarded as an act proper to the vessels, and it was doubtful whether the endocardium had any vessels, it was naturally a rather perplexing question whether the apparently inflammatory effects of endocarditis were really due to inflammation. The difficulty was much greater in the case of the endocardium than in that of other evascular structures, such as the cornea, or cartilage, which produce little or no inflammatory products, because it was easy to deny the existence of inflammation in the apparent absence of its results. But a great quantity of lymph-like deposit was seen in endocarditis, and hence arose a rather keen discussion, some being disposed to think that this deposit was really formed from the endocardium as lymph is produced by other inflamed serous surfaces, while others thought that the apparent lymph was only fibrin from the blood, which had precipitated itself, especially as fibrin was known to be in excess in rheumatism, the disease that commonly causes endocarditis. The following out of this question by Lee and others led to some interesting experiments on the inflammability of the inner vascular surfaces, and it was proved by them that the endarterium will not produce lymph and pus freely on its free surface like the ordinary serous membranes.

But the great number of more careful microscopic examinations which have been made of late have settled the question decisively, although with some alteration of the standpoint from which it is viewed. This alteration arises from the fact that inflammation is no longer regarded as *exclusively* the act of the vessels, although the actions in the vessels compose the chief and most obvious phenomena of ordinary inflammations. It was clearly shown by Virchow and others that a more constant, and perhaps the essential, act in inflammation is one of irritation in the parenchymatous parts of the texture as represented by their cellular elements. With this belief it has become no longer possible for any to hold the opinion that endocarditis cannot occur on the ground that the valves are evascular, while it is further proved that some at least of the valves, certainly the mitral, do contain capillary vessels which have been observed to be congested in inflammation.

Then at present we are able to follow out the inquiry into the process of endocarditis on the same terms as we examine the inflammation of other tissues, so far as the structure of the endocardium is concerned. But it remains true that its circumstances are very peculiar in that it exposes its large surface to contact with the blood. For the living blood is proved by observations and experiments to be always ready to deposit fibrin on a roughened surface, and especially so when the fibrin is in excess in the blood, and when the blood is arterial, and when the blood is in contact with a surface of no vitality, or low vitality, and when its current is checked. Now, all these conditions are found in the inequalities of an inflamed endocardial surface, and especially when the fibrinous deposit has already commenced on several adjacent spots. For there is then a rough surface of low vitality retarding the current by its inequalities; the blood from inflammation being hyperinotic, and the left heart containing arterial blood. Recognising these conditions, one is prepared to believe that any change in the endocardium, inducing a roughening of its surface by swelling or exudation, would soon lead to the deposit of concretions on the rough parts. Such a deposition undoubtedly occurs, and it is this lodgment of fibrin in quantities on the inflamed surface that constitutes the peculiarity of endocarditis, and causes it to differ from inflammation in all other parts.

*Special characters of endocarditis.*—Endocarditis is either plastic or ulcerative. Let us consider, first, the characters of its plastic form. When found in its earliest stages it appears as a slight swelling, with sometimes a pink colour of the membrane; as to this colour, we are not certain whether it can be regarded as due to congestion or imbibition of colouring matter. This always occurs near the edges of a valve in the formation of a row of little elevations along the contact line of its segments, where the friction is greatest. Some have thought that this is due to a peculiarity in the composition of this contact line of the valves. Now, it is true that this line is usually more fibrous and thick than the rest of the valve, but the fibrous thickness itself is clearly due to the chronic irritation of the line by the action of the heart. For it is not found in young subjects and is thicker as age increases; and it is not found in the valves of a normal right heart; moreover, it is found in the valves of the right heart when that heart is hypertrophied. This line of little elevations is what we find in chorea, in acute rheumatism, in puerperal pyæmia, &c., that is, generally in acute endocarditis, when the change in the heart is quite early. Such a change may give rise to a soft bruit in the action of the heart, but it cannot much obstruct its orifices, nor can it poison the blood with its products, so that at this stage it is of little importance. It is only its after consequences that are grave. We have already pointed to the absence of counter-pressure as causing permanence of the swelling of the valve; hence it is that the heart after acute rheumatism, chorea, &c., exerts its strain on an unrecovered, thickened, and softened structure. It is curious to observe how constantly we find in all cases of endocarditis from chorea, acute rheumatism, pyæmia, &c., that the change in the valves is limited to this line of bead-like elevations along the meeting edges of the segments. If a valve with these nodules be cut for the microscope across the plane of its curtain, so as to show a section down through one of the small nodules, this will be found to be composed of a simple cloudy swelling of the tissue of the valve through a multiplication of the cellular elements in its fibrous structure, which here and there by its excess raises the surface into a little hillock. If the hillock takes the form of a distinct projecting grain we always find on the top of it a cap of fibrin separated from its substance by a line which the microscope defines very clearly. This cap of fibrin differs in composition from the hillock itself, though the difference is more easily seen than described, for the organization in both is very low; but the fibrin is almost structureless, while the hillock of swollen valve-substance shows the regularly placed nuclei of fibrous tissue.

Specimens of endocarditis in this early stage are frequent enough, but it is not easy to say what occurs next in the process, because we do not have many opportunities of seeing the intermediate conditions between this which is found in cases of death from the acute disease which causes the endocarditis, and that advanced state of change in the valves which long afterwards proves fatal by disabling them.

It appears to us that in the interval between acute endocarditis of rheumatic fever and the death long after from valvular disease of the heart many frequent repetitions of the inflammation must occur; sometimes we find inflammatory products of two distinct dates on the valves, some recent, some older. But, more usually, a constant state of inflammatory irritation persists, slowly changing the valve. The cause is probably this—that the valve remains swollen through absence of the pressure on it which is required to restore it, as we have already said; and being thus unable to return to its proper size, while it is still softer than natural, it is both subjected to more friction and less able to resist this effect of the constant action of the heart; hence there is a chronic irritation of the unhealed valve, which gradually leads to great thickening of it, so that a scar-like tissue results, in which calcareous salts are often deposited, the whole causing those disastrous effects in contraction and deformity with which we are all too familiar.

The *ulcerative* form of acute endocarditis always begins in a valve. It must not be supposed usually to accompany the milder plastic form we have just described. Some authorities proceed in their description of endocarditis as if when the swelling of the membrane occurs the next thing usually is for this swelling to break down into an ulcer; but we must not let such an impression mislead us into thinking that ulcerative endocarditis is an ordinary part of common cases of endocarditis, such as we meet with in chorea and rheumatism. The occurrence of ulceration is a rare and formidable complication of plastic endocarditis.

We have already described the process of ulceration in the muscular substance of the heart. When limited to the endocardium, *i. e.* when it is only in a valve, an abrasion of the inflamed surface forms and the affected side of the valve suffers a breach. The valve is composed of two layers of endocardium with some fibrous tissue and a few vessels between them. Now, when one layer of the endocardium is breached by the ulcer the force of the heart drives the blood into the hollow and presses before it the remaining layer of the endocardium, thus forming an acute aneurysm of the valve. Such an aneurysm, of course, projects away in the direction of greatest pressure, so that in the mitral valve it bulges up into the auricle, in the aortic valves downward into the ventricle. It is but too easy then for the ulceration or the heart's action to work through the remaining layer of endocardium and so perforate the valve; such perforations are not very infrequent either in the mitral or aortic valves; they are always covered all round with "vegetations," *i. e.* nodular masses of fibrin which hide the opening. These vegetations may reach a large size and become calcareous, and by friction or infection start ulceration in the wall of the heart where they come in contact with it. Such ulcers and perforations of the valves, with the ulcerations of the muscle of the heart which are apt to extend from them, constitute a dangerous disease; its characters are quite distinct from the plastic form of endocarditis, which is only dangerous through its subsequent effects in causing contraction of the valve and so inducing dropsy, &c. Ulcerative endocarditis is always accompanied by pyrexia; it may produce pyæmic suppurations by embolism of distant organs with particles from the ulcer; or the large fibrinous masses around the ulcer may become detached and plug the cerebral or femoral arteries, &c., causing hemiplegia, gangrene of foot, or other severe lesions, through simple obstruction. Ulceration may supervene on chronic plastic endocarditis and its symptoms be complicated with the obstructive effects of this; but it usually kills without dropsy.

In the majority of cases of ulceration a chronic disease of the aortic or mitral valves has existed, and amongst the vegetations micrococci have been found, as well as in the visceral infarctions, showing the infective nature of the complaint. Dr. Goodhart has observed that at Guy's it occurs in groups, and therefore prevails at particular times; some have maintained that it has a malarious origin. These facts have given rise to an opinion that there is a malignant endocarditis in distinction to a simple one, the former being asssciated with septic organisms. Inoculation of animals with the stuff from the vegetations has been unsuccessful. Dr. Wooldridge says that chemical changes in the blood itself are sufficient to produce profound alterations in the lining membrane of the heart and vessels. Some forms of fibrinogen attack the living tissue.

Some say that pus may be found, as little abscesses, in the tissue of the valves under these circumstances, but of this we have no experience. We have, however, already given examples where pus in quantity was found in the heart's muscular walls when the process of ulceration extended from the endocardium to the tissue beneath.

*Limitation of endocarditis to the valves and their neighbourhood.*—We would repeat that all we have said as to acute endocarditis applies almost solely to the valves, and to such extensions from the valvular changes as arise through the spread of the ulcers to the attachments of the valve, or the friction of masses of fibrin on the neighbouring parts. Very occasionally we find a patch of nodular thickening of the endocardium in the left auricle, about the root of the mitral; and once or twice, under exceptional circumstances, as once over a partial rupture of the septum ventriculorum, we have seen inflammatory granulations form on the endocardium remote from a valve.

*Chronic endocarditis.*—We here again allude to those white fibrous patches about the interior of the left ventricle, generally near its apex, which we have already described as disease of the muscle of the heart, because in cutting into them we find they dip deeply into and even through the muscle and extend in it without reaching the inner surface at some parts; so that they evidently belong primarily to the muscle, not to the endocardium, which is smooth and depressed over them. We have mentioned the causation of aneurysm of the heart by these patches.

A few cases have been observed where uric acid has been found in the vegetations, suggesting the term "gouty endocarditis." In a case of xanthelasma Dr. Fagge found some yellow patches on the internal surface of the left auricle.

**Changes in the Forms of the Valves.**—We must now consider the changes in the valves which affect their form and efficiency. These make up a set of conditions of vast importance quite independent of the causes which may have produced them. When the valves are seriously altered in shape they are liable to give rise to two distinct kinds of ill effect, first, in *obstructing* by their contraction or rigidity the opening they guard; and, second, in *allowing a reflux* of blood, which they are placed to prevent. In either of these ways they produce obstruction to the blood, or *ischœmia*, with its general consequences. The principal changes in the valves are as follows:

*Spontaneous rupture of the valves.*—The question has been much discussed whether the valves can be ruptured through violent action of the healthy heart. It cannot be denied that such a rupture may occur, although the accident is rare. It is common enough to have a history of great cardiac disturbance immediately after some violent exertion, when it is suggested that some mischief must have been done to the organ at that time, but we usually know nothing of the state of the valves previously, and the evidence after death of such injury is slight, because the sufferer does not die at once. He lives some time and the valve inflames and the heart dilates and hypertrophies, so that what we find is a broken valve and an altered heart, and it is then left for us to infer which of these was anterior in time. Yet good authorities, as Dr. Quain and Dr. Peacock, relate cases where, after some violent strain, as lifting, all the signs of valvular disease came on. In some of these, and mostly in those we have met with, there was a history of direct violence, and this again throws more obscurity on the cause. In the case of a young man who died in the hospital three days after an injury one of the aortic valves was torn through by a transverse slit. In another case, where a weight fell on a man's chest, a rupture was found on the posterior part of the aorta. As there was no direct injury to the heart, it was thought that the sudden tension of the aorta was sufficient to cause the rupture. And yet this is difficult to understand, for when we reflect on the mechanism of the heart as concerns its valves it will be seen that when it is in its natural form and healthy there would be no strain from the muscle tending to break its valves, for the strain on the aortic valves occurs only in the recoil of the aortic wall, which can hardly be supposed to be capable of splitting the valve; again, the strain on the mitral is relieved by the open aorta, into which the blood can freely flow. But if the heart be at all dilated then comes a strong strain on the mitral columns, which now are not of sufficient length to extend across the enlarged cavity of the heart. Under this strain they will be liable to snap through. This occurrence, namely, the snapping of an overstrained mitral tendon in a dilated heart, we believe to be a relatively very common cause of severe heart disease, converting the very bearable trouble of a moderate dilatation into a hopeless disablement. We have repeatedly traced this occurrence in the history of cases under our own observation. It happens usually either in young rheumatic subjects or in elderly people with dilated hearts due to gout or Bright's disease. The person has been subject to some palpitation and other signs of cardiac dilatation, or he has had rheumatic fever, but, perhaps, suddenly during exertion he is seized with severe cardiac symptoms, which continue until his death a few months afterwards. We then find some of the mitral cords severed, and sometimes very curiously twisted up into spiral knots surrounded by masses of vegetations, and, perhaps, causing ulceration of the endocardium by the pressure of these masses, which are very commonly found evidently dividing the neighbouring cords. We believe the division of the mitral cords may result from this simple cause, but such rupture does not occur in a healthy heart. However we may think the disease arises, the class of cases where mitral cords are divided and the left ventricle is dilated forms one of the most typical groups of the cases known, clinically, as "*regurgitant mitral*," and distinguished from "*obstructed mitral*," which latter cases always have another kind of origin, as we shall presently see. Dr. Allbutt has drawn attention, in an able paper in 'St. George's Hospital Reports,' to some of the consequences of dilatation of the heart during exertion.

*Retroversion of the valves.*—Again, we know that the valves are not made to meet at their edges, but their borders are turned up and meet in such a way that they mutually support each other in the action of the valve. But we often find that the borders of the valves have been bent back by the blood stream which they are placed to resist, so that the current they should check pushes their edges along with it, and escapes through between them. This condition is of less special importance in the mitral valve, where it is a mere accidental complication of more severe disorder already disabling the valve; for the structure of the valve, with its numerous cords holding every part of the surface below, prevents such a retroversion unless the cords are divided by disease. But in the case of the aortic valves we have often found such a bending back of the valve to have suddenly made a very strong man into a hopeless invalid; in this way—the strain of the heart somewhat widens the aortic orifice, so that the valve segments no longer sufficiently meet by their borders and support each other. This widening of the orifice is due to an inflammatory softening of the aortic coats, such as induces aneurysm elsewhere. The valves not supporting each other in some unusual exertion, one of them bends down into the ventricle, and regurgitation is thus allowed. The same result may happen through inflammatory contraction and shortening of the valves, so that they will not well meet; regurgitation being free, and no obstruction existing, these form the very worst class of cases. Such regurgitation usually occurs in strong male subjects whose occupation is laborious, though it is occasionally met with in women who have been subjected to the same conditions. Sometimes the case is not so simple, for the failure of the valves to meet may be due to an actual aneurysm forming about the root of a segment either above or below its attachment.

Retroversion of an aortic valve, as well as rupture of the mitral cords, may result as a consequence of endocardial ulceration. But the chief interest of these states lies in their occurrence as independent and recognisable original causes of grave heart disease. When they occur in the course of ulceration, however, they seriously add to the peril and suffering.

The existence of such ruptures and retroversions affords another instance to show that the muscular force of the heart is the main cause of injury to its structures.

*Deformity of the valves from ulceration.*—We have described the effects of ulceration on the structure of the valves. Its effect on their figure and function is generally very destructive of the normal state of both. During the activity of the ulceration there are always present large masses of vegetation attached about the inflamed spots, and these obstruct the opening protected by the valve, while they also prevent its efficient closure, so that in ulcerative endocarditis both obstruction and regurgitation may be present at the affected orifice. But the obstruction, if there be no antecedent chronic mischief, does not reach such a pitch as to cause any considerable dropsy, while those other grave results of the ulcer which we have already described will often destroy life before there has been much time for the slower production of contraction, &c., that would follow if the ulceration were healed. Obstruction is generally most marked in these cases at the aortic orifice, which is more easily closed by a mass of vegetations than the mitral. Regurgitation is, on the other hand, often very considerable in these cases, especially through the mitral when its cords are divided. Ulcerative endocarditis may heal, leaving the valves much reduced by the destruction caused. Thus, we have seen a case where a large part of two aortic segments had been removed, and one where the aortic valves were reduced to relics.

*Deformity from contraction, "ossification," &c.*—Contraction of the valves is generally the effect of that prolonged irritation which is the natural sequel of their acute inflammation; it is by far more common in the mitral valve than in the aortic. But the aortic valves do not escape in all cases; thus, we see the obstructive effects of inflammation of the aortic valves in two of them becoming adherent, although their efficiency may remain. If this union be perfect, the partition between them is in time dissolved, and the two valves become one, or an adhesion may take place between the edge of one and the aorta, and thus one valve becomes two. But in the mitral valve, owing to endocarditis, a thickening of all the parts included in the structure frequently occurs. Thus the curtains become three or four times their natural thickness, and form hard masses like cartilage; indeed, we have microscopic sections from specimens showing well-marked cartilage in such a thickened mitral; the orifice is altered in shape, becomes round, and will scarcely admit the point of the finger; or more commonly it has the form of a button hole and may be no larger than a shirt-button hole; at the same time the tendinous cords become drawn up, shortened, and thickened, and sometimes several unite together into a thick tendinous mass. We have met with examples where all the cords had gathered into two short pillars of white fibre, one at each end of the mitral opening, which was half an inch long and one sixth of an inch broad, the whole structure looking like a little funnel composed of tendinous tissue. The endocardium on the septum of the ventricles may be much thickened close to the valves, and sometimes we meet with a very remarkable thick band, projecting into the cavity, as though it helped to maintain the column of blood, and so compensate for insufficiency in the valves; sometimes there are several smaller pieces, which may curiously resemble valves in their appearance.

Although there may be good evidence of previous endocarditis to account for this thickening and contraction of the mitral valve, it is remarkable that in many cases such proof is altogether wanting, and the existence of the stenosis may be traced back to childhood. Dr. Fagge, who took the trouble to inquire into the history of a large number of cases, came to the conclusion that stenosis of the mitral valve was congenital in the majority of instances, and that it was much more common in the female than in the male.

But before coming to this conclusion we must remember that all theories of its production have gone on the supposition that valvular disease must have had an acute origin, and that this was probably of a rheumatic kind. It may, however, in many instances be of an essentially chronic nature, for just as there is a chronic inflammation of a joint of a rheumatic or gouty nature, so there may be a chronic endocarditis accompanying it. We may remember also, if the inflammation of a valve have an acute origin, the subsequent thickening and malformation is of a slow and essentially chronic kind. How constantly does it happen that during an attack of rheumatism no evidence is present of an endocarditis, and yet in the course of a few weeks or months a bruit proves that a change in the valve has been going on since that time.

Dr. Pitt had also examined into these cases, and found that when stenosis is met with in adults one fourth of these at least are the subjects of Bright's disease. Dr. Goodhart had previously observed the same fact.

*Stenosis of the tricuspid valves* is sometimes met with. It is usually associated with a similar condition of the mitral valve, and is much more frequent in women than in men.

In valves which have been long diseased calcareous changes may take place. A very favourite seat is the inner side of the mitral valve, between it and the aortic; but sometimes the whole mitral orifice is surrounded by a bony ring. In the aortic the deposits generally begin at the bottom of the valves, at their place of attachment; they are often large, so that at the post-mortem examination the finger will feel their presence at a time when they are not yet visible.

This calcification is the most frequent cause of simple aortic obstruction. The earthy concretions grow into great masses that invade the valves until at last they are converted into immoveable nodular masses of earthy material, with the orifice of the aorta reduced to a little chink. Such examples we may sometimes meet as causes of the sudden death of hale-looking old men who never had dropsy, and sometimes they are found in the bodies of old men who have died of independent diseases, even when the obstruction of the aorta is nearly absolute; such cases are very surprising, and prove how comparatively innocent is simple obstruction of the orifices.

*Fenestration of the valves.*—There are still other changes found in the valves. We will first allude to the so-called fenestration of the valves. We refer, especially, to what may every day be met with in aortic and pulmonary sigmoids; that is, a perforated or fenestrated condition of them above the crescentic line where they are apposed. The perforation can, therefore, produce little ill effect so long as the free border keeps its firmness and continuity so as to do its share in resisting tension. These small holes have often been looked upon as a result of atrophy, but there is little proof of this; we have seen them in young people, and we have always regarded them as congenital, for in some of the lower animals the sigmoid valves are attached to the artery by thin tendinous cords (which are produced here by the perforated condition) in the same way as the auriculo-ventricular. This may be seen in the heart of the shark.

*True aneurysm of the valves* is sometimes met with. Thus, in the mitral valve, pouches may be seen opening towards the ventricular side: these are sacculated. There is also an aneurysmal dilatation of the whole valve, as may be seen in the mitral, where both curtains bulge out into the ventricle, or in the aortic valve, which becomes greatly distended.

*Malformations of the valves.*—We mention these last, for as, no doubt, they arise from inflammatory changes and adhesions, we shall be prepared to know how they are produced. Thus, a funnel-shaped membrane, taking the place of the pulmonary valves, is evidently formed by their union, for the lines of junction may still remain. This was the case in a girl who died in the hospital only a short time ago, and who never had any previous illness during her lifetime, and, therefore, there was every probability that the disease occurred in fœtal life; such cases of conical pulmonary valve are not very rare, and the subjects of such disease may reach elderly life. With this conical membrane at the pulmonary orifice we may contrast the condition of the aortic valves in the same heart, and see what has occurred there from inflammatory adhesion; instead of all three becoming united, two only have done so, and in course of time the partition has been dissipated, and the two have become one, and thus we account for a *deficiency of valves.* There can be no doubt whatever that this is the way in which these so-called malformations come about, for we think the same thing may occur in adult life as a result of inflammatory adhesion. We have, for example, four hearts: in one we see two valves united as far as their middle, but yet the two free edges can be seen; in one we see a perfect blending of the adherent sides, and the united structure has sunk down below the level of the valves; in the third specimen we might imagine we were still looking at the same at a further stage, for it exactly resembles it, only the uniting partition is much less, and scarcely divides the large valve into two; while in this fourth heart we see a large valve with only a trace of partition at its bottom, and thus two valves have been formed out of three. We have in the Museum a pulmonary artery with two, and a good specimen of aorta with only two valves. *Supernumerary valves* are often formed in like manner. Thus, owing to an inflammatory adhesion, the free edge of the valve becomes fixed to the aorta, and thus two valves made out of one, producing four in all. This supernumerary one is much smaller than the others. Where the valves present quite a healthy appearance, and are uniform in size, it might be a question whether the excess arises in the way mentioned; but since four valves are much more frequent in the pulmonary artery than aorta, and as the former appears more susceptible of disease in uterine life than the latter, the regularity of the valves may be owing merely to the circumstance of the early period at which the adhesion takes place, *i. e.*

when all the parts are plastic. Such perfect pulmonary and aortic valves may be seen in our specimens. In the heart of the girl we have just spoken of, with deformed aortic and pulmonary valves, it will be seen also that the mitral is malformed : this is rare. We have twice seen the front segment of the mitral connected to the septum of the heart near the apex by a long, worm-like, fleshy band, and in one instance two mitral valves.

**Valvular Obstruction and Regurgitation.**—We believe that aortic obstruction without regurgitation is a rare form of heart disease, and that those authors who make it the most common are in error. Both aortic and mitral obstructions are generally the results of chronic inflammation, or of a calcareous degeneration, which may or may not be the consequence of inflammation. The distinction between the effects of regurgitation and obstruction is important; regurgitant disease is more formidable than obstructive. It is true that in a large proportion of cases both regurgitation and obstruction occur, but the effect on the heart will differ much according as the obstruction or the regurgitation prevails in the case. This may be stated as the rule—that the cavity of the heart behind the affected valve hypertrophies in proportion to the obstruction and dilates in proportion to the regurgitation at that valve, a very important distinction, since the danger and suffering are in proportion to the dilatation only. We may say, then, that obstruction with its attendant hypertrophy is less formidable, while regurgitation with its attendant dilatation is more formidable. As to the relative danger of mitral and aortic disease generally, so much depends upon whether the disease is obstructive or regurgitant, and whether it is combined with ulceration, that we do not think any useful rule can be given. But as the aortic valve is more subject to retroversion, which allows a large unobstructed regurgitation, and therefore a great dilatation, the immediate dangers of aortic disease are greater. As a matter of experience we should say that aortic valvular disease brings with it ten times more danger of sudden death than mitral disease, and this is due to the ready retroversion of the valvular segments.

**"Polypi"—Thrombosis or Ante-mortem Coagula in the Heart.**—A dilated heart was at one time called an aneurysm of the heart, a very erroneous expression, although in one respect they may behave alike, and that is, in the occurrence of coagulation of the blood within them. The coagulation is from the same cause in both instances, viz. stagnation dependent on the small proportion which the orifice bears to the dilated cavity. We have already drawn attention to the deposition of fibrin on the inflamed endocardium, making the great mass of vegetations in endocarditis. At that time we spoke of the conditions that favour such deposition of fibrin. The chief of these, however, are two—an uneven surface and stagnation of the current.

To understand the common appearance of ante-mortem clots in the heart we must recall the appearance within its apex, &c., and remember the many recesses between the columnæ carneæ, many of which recesses form shut sacs. The existence of these would easily appear to be a constantly threatening source of danger from coagulation, but that in the systole of the natural heart they are closed completely and all the blood expelled from them. When, however, the heart is much dilated, these recesses in its wall do not entirely close in the systole, but remain as permanently patulous cavities, in which blood, if the general current is very slow, may be entirely motionless, and so be ready to coagulate. In this way it is that clots form in the recesses of the columnæ carneæ near the apex or appendix of the dilated cavity of the heart, especially the left ventricle and the right auricle. Once formed, the surface of the clot receives additions of fibrin layer by layer as any other unnatural surface in the blood stream always does, so that at last there is a considerable mass with a laminated structure outside, but containing some blood in its interior. We must distinguish the first coagulation of the entire blood from the deposition of colourless fibrin upon it. This process takes place in several neighbouring recesses at the same time, and some of the masses, as they grow out of their recesses, meet together and coalesce, and thus are formed greater or smaller laminated bodies. The laminæ do not cover the whole surface concentrically, but each is only of partial extent, so that their edges show as sinuous lines upon the "polypus," as such a concretion is called when it projects into a cavity of the heart. It gets this name, "polypus," because of its resemblance to polypoid growths in mucous surfaces, being always rounded pretty smoothly on its free surface where subjected to the current, though it has root-like projections extending into the recesses it first formed in.

But all polypi are not so small in their beginnings. Some form by a more extensive coagulation of the blood in the apex or appendix, the clot so formed implicating the blood in the recesses around, so as to be held in its place by its ramifications among them. Over this clot layers of fibrin form as over the smaller ones, and then the look of those that arose very slowly and those that come more suddenly is very much alike. But the difference is in their interior. For all the while these polypi exist they are undergoing degenerative changes, which result in the softening down of their central parts to a pulp of creamy consistence. Now, in the slowly formed concretions, composed chiefly of fibrin, the creamy matter is white, but in the more hastily formed larger blood-clots the cream is coloured with the more or less decaying blood pigment, so as to be purple or brownish-purple. By the amount of blood pigment in the interior of these clots we may generally judge the size of the blood-clot which they were originally founded upon, although, no doubt, some amount of decolorisation of the clot is always going on, the red corpuscles breaking up, and the *liquor sanguinis* around washing out the pigment they leave. It is true that the small polypi contain least pigment, as if most thoroughly washed, but these are they that grow very slowly from deposit of colourless fibrin.

The action of the blood on the original large clot is much impeded by the layers of fibrin that surround it, so that the process of decolorisation proceeds but slowly. If we examine the contents of the softened polypus microscopically we find it chiefly composed of detritus of fibrin in an amorphous state, but it is not right to say that the microscope always proves that these concretions are not purulent, for sometimes we have found a great number of pus-like corpuscles in them which we think, perhaps, are due to the wandering of white corpuscles from the blood through the fibrin layer into the interior of the polypus. These polypi are not without their special dangers. They may be dislodged and pass on to obstruct the large arteries, or become entangled in a valve. Or a polypus may burst and throw its contents into the blood stream, leading to embolism. We have never seen decided examples of this, but cases are recorded. It is said by some that the result of this occurrence would be suppuration, &c., in the part embolised; we should not, however, expect this, since passively formed fibrin is inert in the tissue it reaches, and it is only when the fibrin comes from a seat of acute inflammation that it sets up inflammation.

A hollow, collapsed, fibrin-concretion is very like a polyp or sea anemone. It is a very strange-looking pathological object, although its explanation is so simple. Before Goodsir pointed out the passive nature of the process forming it, its pus-like contents naturally led to the idea that it was a purulent sac in the heart, and therefore of a very serious nature; but the almost constant connection of it with cardiac dilatation rather than with suppurative disease might have suggested its true character. However, it must not be assumed that it is always devoid of active powers. We have on two occasions found these polypi in the heart in pyæmia, and have several times met with suppurative thrombus (*i. e.* pus in the veins) in the places where great venous stagnation occurs, as in the pelvic plexus, &c. Again, the polypi do often, as we have said, contain a great many pus-like corpuscles, so that we do not think their origin can be held to be always necessarily passive. Remember, too, that cancer may spread in them. Rindfleisch speaks of their outer layers being better nourished than the inner, which assumes some activity in them, although he does not believe in the spread of cancer in them, and takes their nature to be passive. We think there is evidence that such polypi may have active as well as passive causes—in other words, that the state of the blood itself is in part a cause of their formation.

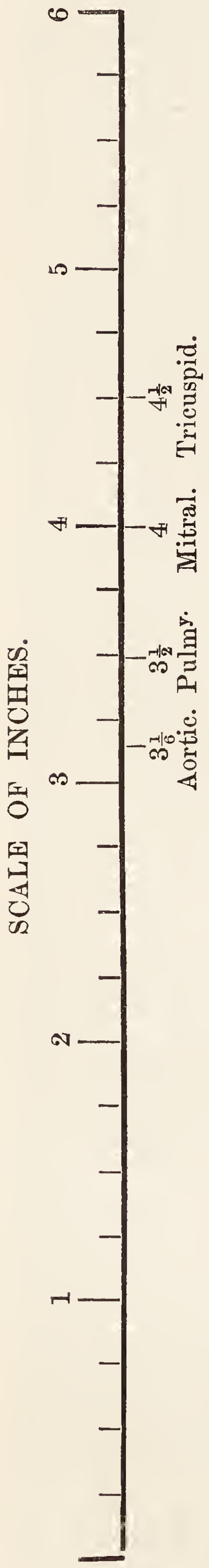
A coagulum may form in the apex of the heart, and, being dislodged, be cast either into the pulmonary artery from the right heart, or into the aortic system from the left. It may then give rise to further coagulation or thrombosis.

*The blood in the heart after death.*—It must be remembered that the right side of the heart, and the vessels leading from and thereto contain naturally, after death, a coagulum such as would form in a basin during venesection. Thus, as the body lies on its back, the red corpuscles sink down and the fibrinous clot forms above them, and thus in right auricle and ventricle it is found, white or buff anteriorly, and red posteriorly; and connected with these clots in the auricle are those proceeding from the vena cava and jugular vein, and from the pulmonary artery with its branches; the shape of the valves being modelled upon the latter. On the left side the blood is less firmly coagulated, the auricle being filled with a dark semifluid blood, and the ventricle with a small quantity of the same, any coagulum found within it being very small. This is on account of the usually contracted state of the left ventricle. Also, it is more rare to be able to detach branching coagula from pulmonary veins. Why the blood should be dark and scarcely coagulated on the left side is not quite explained; but it must be remembered that the left heart lies low, so that it receives the corpuscles while the clear fibrin is in the arch of the aorta, as that lies on a higher level. In diseased hearts with dilated left ventricle we may find a large softish clot, but seldom so well formed as on the right side. If we discover anything unusual in these clots, we shall more rightly regard them as the consequence of a peculiar mode of death than as the cause of it. If a clot forms before death, it can generally be told from the fact of its being firm, adherent to the walls, and having lost its colour, also, when opened, it is softened in the centre; whereas we always find some evidence of the gravitation of the red corpuscles in clots formed after death, so that they are pale on the upper side and dark on the lower. A little experience will enable us to distinguish. By the same means we shall be able to distinguish an old clot from a recent one in a small vessel, as the cerebral to which we have already alluded; but it is not always easy to say whether a fibrinous concretion has been formed in a vessel, or whether it has been carried from a distant part. If it be supposed to have been brought from the heart, it must be compared with the vegetations there found. Its shape, and the mode in which it is placed in the vessel, will often show whether it has been formed there or not; for if coagulation occur from a disease in the tissue, all the vessels leading to it, up to a certain sized branch, will be found filled; whereas, if the fibrinous plug be carried from a distant part, it will lodge at that spot where its progress is arrested, and the smaller vessel beyond will of necessity be found empty. Under the headings of Kidney and Spleen we shall have occasion to speak of fibrinous masses found in them in connection with heart disease, which are due to some portions of vegetations carried from the valves; they are more frequently found in connection with such vegetations than not, but very often in cardiac disease without such vegetations, and, therefore, the theory in this case is not without doubt in some minds.

A very interesting class of cases connected with this subject was mentioned by Simpson, in which during the puerperal state there is a disposition for clots to appear in the femoral arteries, so that gangrene of an extremity sometimes takes place, or in the pulmonary artery, causing sudden death; and hemiplegia also sometimes occurs. We believe this class belongs to the sudden thrombosis of the ventricles, and embolism, which we have just described.

The effects of congestion on the various organs in valvular disease will be treated of in the successive chapters — such as the apoplectic condition of the lung, the nutmeg liver, indurated spleen, and hard large kidneys, as well as the intensely injected state of the stomach and intestines.

The table of measurements which hangs in the post-mortem room we have exchanged for the old one of Bouillaud. The measurements are taken from a great many examples; and we have given them in inches instead of in centimetres, as by Peacock and others; they are in consequence, perhaps, not quite so accurate, but students will be better enabled to carry them in their memory.



MEASUREMENTS OF HEART.

| | |
|---|---|
| Circumference at base . . . . | 9 in. or rather more |
| Length . . . . . . . . . . | rather more than breadth |
| Circumference of right ventricle . | 5 in., nearly |
| Length of right ventricle . . . | 3¾ in., about |
| Thickness of walls of left ventricle | ½ in., about |
| ,, ,, right ventricle | ⅙ in., about |
| ,, septum ventriculorum | intermediate between two above |
| ,, left auricle . . . . | ⅛ in., about |
| ,, right auricle . . . | rather less than left |
| Orifice: left auri.-ventric. (mitral) . | 4 in. in circumference |
| ,, right ,, (tricuspid) . | 4½ in., or rather more |
| ,, aortic . . . . . . | 3⅙ in. |
| ,, pulmonary . . . . . | 3½ in. |

It will be seen that the tricuspid orifice is larger than the mitral, the mitral than the pulmonary, and the pulmonary than the aortic. The pulmonary is larger than the aortic by ⅛ part, and the mitral larger than the aortic almost by ¼ part (being ¾ inch larger), and the tricuspid is half as large again as the aortic.

These measurements were made by a graduated cone, which being inserted into the orifice, the line at which it is stopped is read off as the size of the opening; when there is much rigidity, and the valves have lost their pliancy, the instrument is useless; we must then lay open the orifice and measure across by a graduated tape, taking care to fit it into all the inequalities.

As regards the capacity of the cavities, each probably contains about three ounces of blood; but it is by no means certain, as some maintain, that the right is more capacious than the left; it is said, too, that the ventricles hold more than the auricles. The weight of the heart is about nine and a half ounces; this is the average taken from all our cases during three years; as these are mostly cases of chronic disease, and the manifestly enlarged hearts are excluded, it is probable that in healthy persons the weight may be somewhat higher than this. In wasting diseases it weighs sometimes as low as four or five ounces; or in hypertrophy may reach to two and a half pounds; in the latter cases, the wall of the left ventricle may measure an inch or more in thickness, and the septum participates in the changes of the ventricles.

# DISEASES OF THE ARTERIES

**Malformation.**—Irregularities of distribution concern principally the anatomist, and are treated of in anatomical works. We have already mentioned the most important malformations of the great vessels when describing those of the heart, to which they properly belong; we would especially speak here of the congenital contraction of the aorta, and its constant position at the entrance of Botalli's duct, many examples of which may be seen in the Museum.

**Hypertrophy.**—The arteries are very capable of enlargement, as we see in the increased size of collateral vessels after ligature of a main trunk, or in the gravid uterus; we find, though, that in such cases the arterial walls are thin and vein-like. Thus, the enormous cervical and humeral arteries communicating between the arch of the aorta and its thoracic portion in a case of congenital contraction of the aorta at the junction of Botalli's duct were much more like veins in the structure of their walls. The arteries are thick in elderly people, especially when the subjects of granular disease of the kidneys. This thickness is chiefly due to hypertrophy, but the coats are usually in a degenerated condition. There has been keen discussion about the state of the arteries in cases of granular kidney, Dr. Geo. Johnson maintaining that the arteries of the body have their muscular coat greatly hypertrophied, while Sir W. Gull and Dr. Sutton assert that they are thickened by a fibroid change in the outer coat especially, which these authors term "arterio-capillary fibrosis." The latter regard the change as from the outset pathological, while the former sees in the contraction and consequent hypertrophy of the arterial muscle a physiological and conservative process designed to prevent the access of noxious substances to the tissues. We have found that this hyaline and muscular thickening of the arterial walls does occur, but any considerable thickening is accompanied by degeneration of the coats, and very rarely is a simple hypertrophy, so that the statement of both observers is correct. The term hyaline is used to designate the adventitious material which is translucent.

There is, however, great hypertrophy of the *renal* arteries in granular disease of the kidney, just as there is hypertrophy of the hepatic arteries in cirrhosis of the liver, and of the bronchial arteries in old fibrous disease of the lung.

**Atrophy.**—It is said by Virchow that in chlorosis the whole vascular system, including the arteries, is in an atrophied condition similar to that which prevails in the blood. This condition has been thought to be congenital in chlorotic persons. We have no special experience on this subject, but the point raised is one of great interest.

The aorta may be very small in cases of obstructive disease preventing free access of blood to the left heart, especially in congenital contraction of pulmonary orifices.

**Inflammation—Arteritis.**—The remarks we made upon the general and comparative nature of endocarditis apply to inflammations of the arteries and veins, since their lining membrane is likewise devoid of vessels. It is true of the arteries, as it is true of the heart, that we do not meet with a general acute inflammation of the interior such as would resemble a general pericarditis or pleurisy in producing lymph over large tracts of surface.

Experiments on blood-vessels have failed in developing those well-marked phenomena of inflammation which are seen in ordinary serous membranes, and thus, in those cases where a vessel is found filled with fibrin, adherent to the walls, it is probable that much of this is due to a mere coagulation of the blood, and not to an exudation from the arterial surface.

*Acute general arteritis.*—It was formerly thought that the deep red staining, which we now know to be due to imbibition of hæmatin through solution of the corpuscles of the blood, was a sign of inflammation; and even when it had been clearly proved that such staining caused a redness, it was still maintained that some redness was of an inflammatory kind. But this belief is now entirely abandoned, and the accounts of acute aortitis given by Peter Frank and others are recognised as erroneous; they were founded on descriptions of the symptoms preceding death in cases where early solution of the corpuscles occurred. We may say that general acute arteritis is unknown.

*Chronic general arteritis.*—Cases are occasionally met with where the great arteries, in young and vigorous subjects, become obstructed, so that no pulsating vessel can be felt during life in any of the limbs, and the arteries are found thickened and containing adherent clots after death. In other cases the same condition may be less general, yet extensively distributed. The character of these cases is not agreed on; but we have found extreme examples of such disease in the arteries to coexist with the fibrous thickenings of myocarditis in the left ventricle, suggesting plainly that the arterial disease is of the same inflammatory nature, and that a general chronic inflammation of the interior of the vascular system was present. In some of these cases the disease has curiously selected corresponding arteries at their corresponding parts. Thus, both the femoral and both the brachial arteries may be thickened and stopped up with clots when no other arteries are implicated. Such a condition brings great danger of gangrene of the limbs in which it occurs. It is fortunately very rare, but must be borne in mind as occasionally met with.

For instance, a girl was at one time in the hospital in whom no pulsating artery could be felt in any part of the body, and this state had been coming on for some years; after death, all the larger trunks were found to be narrowed or obliterated. So many cases have now occurred in young people that the term *obliterative arteritis* has come into use.

*Circumscribed arteritis.*—This may be ulcerative. We need only allude shortly to the extension of neighbouring ulceration to the coats of an artery, though the clinical importance of this dangerous occurrence is very great. It is a frequent cause of death. Thus, for instance, a bubo, or an ulceration beneath the skin extending from a diseased knee-joint, may open the femoral artery, or severe ulceration about the fauces may reach the carotid, or an ulcer on the face open the facial artery. More frequently an ulcerous state of the stump after amputation may eat into the main artery of the limb. Another important and not infrequent example of such ulceration is furnished by the spread of stomach ulcers, so that, through adhesion of the stomach, they reach and open the splenic or some other neighbouring artery. When an ulcer reaches an artery the wall of this becomes thin and brown, and at length the vessel is burst by the pressure within. We have met with an example where, the splenic artery being thus attacked by gastric ulcer, a clot formed within it at the affected spot, but was pushed on as an embolus into the spleen. This occurrence might be regarded as an attempt, though unsuccessful, to close the vessel and avert the danger.

Another method of origin for ulceration of an artery is that which is sometimes found in the spread of ulcerative endocarditis; here we may see an erosion of the wall of the aorta at a point struck by a hard freely moving vegetation on a segment of the aortic valve. Sometimes patches covered with vegetation, and evidently acutely inflamed, but without loss of tissue, are found in the ascending aorta when its valves are affected with ulcerative or other severe inflammation. The great danger of these occurrences lies in their tendency to displace the valve, and so retrovert one or more of its segments.

Next we must notice a degree of inflammation which we will call *inflammatory mollities.* In this there is no ulceration; it leads to softening and swelling of the arterial wall, and so may induce either rupture or aneurysm. It is important to notice that such an inflammation may arise from embolism under a certain defined condition, to which enough attention has not been given. This condition is that the embolus must come from a seat of inflammation; such an inflammatory embolus, reaching and lodging in a smaller artery, such as the femoral, meningeal, or any other, inflames and softens its coat, so that the vessel either bursts or yields to make an aneurysm. Embolism of arteries is very frequent, but it does not often give rise to aneurysm or effusion of blood, because the embolus is not inflammatory, except in rare cases of ulcerative endocarditis. This disease is infective, and is now shown to have specific microbes. The aorta is the most frequent seat of this inflammatory mollities. Here it is generally spontaneous, or, at least, arises in the aortic wall as a proper disease of its tissue.

Inflammatory mollities is the common cause of aneurysm in young subjects, but it is not limited to early years. It may also induce rupture of the aorta, or, by being near the root of an aortic valve, it may allow the valve to turn over. This is one of its greatest dangers in young hard-working men. The affected part of the vessel is generally sharply circumscribed, and when the vessel is opened it bulges inwards, because the swelling of the coat enlarges its extent of surface; prior to the opening of the vessel this part had bulged outwards, and was, in short, already an incipient aneurysm. Such a patch no longer has its proper yellowish opacity, but it is of a bluish moist appearance, and between the fingers it feels flabby and inelastic, more like wet membrane than like aorta, so that one can trace it by the fingers with one's eyes shut. The microscope shows that an inflammatory action is present: there are exudation cells in more or less abundance throughout the wall of the vessel, often in all its coats, and we have seen such cells collected in the outer coat of such an artery into small aggregations like tubercles in appearance. Though we ourselves have never seen anything that we should call "pus" in these arteries, yet this appears to have been noticed by other observers, particularly Mr. Lawson, who stated that phlegmonous inflammation of limited portions of walls of the arteries or veins is by no means rare at the Cape, where it leads to aneurysm or rupture of such vessels. Dr. Charlewood Turner has shown that some of these cases of aortitis are of an infective character, and of the same nature as ulcerative endocarditis. He found that in connection with pyæmia, scarlatina, and other blood diseases, the aorta contained fibrinous coagula adherent to the softened and degenerated walls of the vessel, and in their midst were micrococci. This implication of all the coats, including the adventitia, explains why, as a sequel, the artery comes in these cases to be firmly fastened to parts around it. A contraction of the lymph, as in other inflamed parts, may seriously narrow the calibre of the aorta. We have seen cases of death from this contraction. It is interesting to observe the distribution of this inflammatory mollities in the aorta; it is scattered irregularly throughout in circumscribed patches, reminding one irresistibly of a cutaneous eruption, so that it seems fair to regard the acute forms, at least, as eruptive in their nature, that is, as local actions dependent on a peculiar general state.

Next we must notice a lower kind of inflammation, a *semi-cartilaginous thickening*, chiefly limited to the inner coats. This shows gradations connecting it with the inflammatory mollities we have just described. It appears in the deep layer of the inner coat as a very slow swelling, gradually elevating the surface of the affected part, which at the same time grows tough and inelastic, acquiring very much the same bluish cartilage-like appearance we mentioned as present in mollities, so that the two conditions are apt to be confused together as semi-cartilaginous thickening. The microscopic appearance in this state differs from that in the softer and more acute form in showing more of a hyaline or slightly fibrillated matter in it, with much fewer cells—that is, it differs as slighter inflammations from severer. If we study the borders of the patch we shall see that the hyaline material arises in a swelling of the fenestrated lamellæ of the intima, with a multiplication of its cells.

These patches are always of slow growth, and when seen they show all stages of development together. Of their decay into a fatty paste or atheroma we shall speak presently. They may acquire an indurated condition by slow organisation, if time be allowed them. Their substance is like that of the proper inner arterial coat, and although not perfectly like it, yet they offer in this stage a very beautiful example of the development of inflammatory products to the likeness of the part they were produced in.

*Syphilitic arteritis.*—So many cases of aneurysm have been met with in syphilitic subjects that scarcely a doubt could remain as to their cause. The morbid changes sometimes found in such subjects would lead to the same conclusion, although the appearances are not characteristic, showing nothing more than fibrous patches. With the smaller arteries there is no doubt of a syphilitic inflammation which is characteristic. In a paper on visceral syphilis, published in ‘Guy’s Hospital Reports’ for 1863, mention is made of cerebral arteries containing nodules, with a constriction of their calibre, and they were regarded as syphilitic. Since then Heubner has investigated the subject closely, and he describes, as we had done, vessels with nodules upon them much altered in shape, and the calibre almost obliterated. His observations have been confirmed by all subsequent pathologists, that the growth, consisting mainly of cells with some fibrous tissue, springs up in the intima, leaving the muscular coat and adventitia almost free. A section of the vessel shows a distinct membrane of endothelium on one side of the growth, and the fenestrated membrane with the muscular coat on the other. The inner coat may thus be seen twice as thick as the middle and outer coats together. Although this is the most characteristic form, we often see cases where the nodules and thickening are almost entirely limited to the adventitia; occasionally, even at the base of the brain, we see the vessels matted together, with thrombosis internally, as if all the coats had been involved. Although it is rare to find arterial disease in hereditary syphilis, Dr. Barlow exhibited a typical specimen of it in an infant fifteen weeks old. Dr. d’Ornellas has related, in a French medical journal, the case of a man who had gangrene of the fingers of one hand. It was regarded by the best professors in Paris as due to a syphilitic arteritis.

**Degeneration of the Arterial Walls.**—*Endarteritis deformans.*—This is generally the result of chronic arteritis. Thus, the *fibroid* or *semi-cartilaginous thickening*, as we have already intimated, is rather a state left by the slow arteritis that once caused it than a sign of action present at the period of death. When such processes as we have just described have lasted long in elderly people and spread very extensively throughout the aorta—their chief seat—then the semi-cartilaginous thickenings are found to have undergone retrograde changes, softening them to yellowish pulpy patches, or hardening them to stone; meanwhile, the artery is wide and irregular from partial yielding of weakened spots, so that its interior may be greatly defaced. Extreme examples of this are very remarkable, the roughened aorta widening until it reaches three and a half inches or more in diameter, and its great branches equalling the aorta itself in size, the whole condition well meriting the name *endarteritis deformans* used for it by Virchow.

The retrograde changes in inflamed arteries are of two kinds, called *atheroma* and *calcification.*

*Atheroma* commences in the semi-cartilaginous thickening as a fatty degeneration. We have already seen that this thickening affects the inner coat of the artery, so that it is here that the fatty change begins. The fat appears in the form of grains occupying the spindle-shaped spaces that contain the cells in the tissue; soon the fat quite hides the cells, and at last it accumulates so in their spaces that these break into each other, the intervening tissue wearing away meanwhile; so that at last a considerable soft fat patch is found covered inside by the remains of the internal coat, between which and the middle coat it lies. Chemical changes then occur in the fatty detritus resulting in the production of a quantity of cholesterine, which gives a glistening look to the matter when it is brought to view, so that it appears as a grey, rather glittering paste, which under the microscope shows only fat-grains and fragments of texture and cells with crystals of cholesterine. The accumulation of these products causes the patch to swell beyond its original size, and at length it may burst into the aorta. The blood then enters and washes out its contents. After thus finding a way into the wall behind the inner coat, the blood may be forced along between the coats, producing a dissecting aneurysm. If a fibrinous coagulum then forms, the inner coat being pushed inwards, a complete stoppage of the circulation may occur. In one of our specimens the aorta is so obstructed, causing gangrene of the extremities; and in another there are two distinct obstructions, one above and the other below the diaphragm. In a third there is complete obstruction at the lower end of the aorta; the coats have separated, a fibrinous concretion has taken its place within and between them, and thus a stoppage of the circulation occurred at this spot. In some cases the vessel may rupture and blood break through, causing instant death; and this has also happened, though rarely, in a limb. It is not so common, however, as the blood running between the coats. It was the contemplation of such cases of which we have spoken that first suggested to Sir A. Cooper the possibility of putting a ligature on the aorta.

*Calcification.*—Atheromatous thickening of the sinuses of Valsalva is important in preventing the due closure of the valves, and producing all the effects which are seen in disease of the valves themselves. In connection with disease at this spot the coronary arteries are sometimes quite closed. But, fortunately, all atheromatous patches do not soften down and rupture in the way we have just described. In by far the greater number of the patches the fatty and granular paste soon undergoes a greater and greater addition of lime salts together with disappearance of the fat, until at length a firm plate of bone-like appearance and consistency is found, forming what is called calcification of the artery. It is doubtful whether the fatty degeneration which constitutes atheroma always affects the semi-cartilaginous patch before it is converted into a bony plate. We rather think that in many cases the change from semi-cartilaginous thickening to the bony plate is effected directly by the simple deposition of lime salts in the still firm, half-cartilaginous tissue.

In these hard plates or calcification of arteries of which we have been speaking true bone seldom exists; indeed, in all the cretifications connected with the heart and arteries which we have examined (unlike those of the cerebral and spinal membranes) no true bone has been found. If we take one of these hard plates from the aorta, and grind it down so that it will form a fit object for the microscope, we see no lacunæ or other constituents of bone, but merely a number of round masses, disposed in a matrix of fibrous tissue. It has, however, been found by some observers. In a case under Mr. Howse, where the axillary artery had been violently torn and death occurred in a few weeks, the vessel had become quite bony, and true osseous tissue was found in it. Dr. Coates also says in the senile changes which the vessels undergo we may sometimes find imperfectly formed bone in the middle coat.

The roughness of the inside of the artery, caused by atheromatous and bony changes, may induce coagulation of blood upon it, so that considerable masses of clot may be found clinging or hanging to the surface; such masses of clot may be detached and pass on to obstruct smaller arteries, passing, say, from the aorta to the femoral artery. Such an occurrence is not very uncommon as a cause of senile gangrene.

The three conditions, semi-cartilaginous thickening, atheroma, and calcification, are, then, the successive effects of the low inflammation for which the name *endarteritis deformans* is used by German authors. In our description of them we have hitherto kept in view their appearance as presented in the aorta. The middle-sized and smaller arteries show changes essentially similar, which, however, most usually do not go far in the atheromatous, or reach the ossifying, stage. These changes are especially frequent in the arteries of the brain, spleen, and heart. In the brain they are the common causes of apoplexy and softening; occasionally a diseased coronary artery has been known to burst into the pericardium. The form usually met with is a thickening of the internal coat, having all the characters of the semi-cartilaginous change before described. The thick coat is generally rendered opaque by some degree of fatty degeneration, but is not so fatty as to be softened; indeed, these arteries certainly often become calcareous without a preceding stage of fatty softening. The thick internal coat is usually very easily separated from the others, so that we may take from the inside of such a cerebral artery a tube like a quill, leaving the artery apparently improved by the process, but really now only consisting of the middle and external coat.

While thus comparing these states of the smaller and greater arteries we should draw attention to the fact that the aorta may be exceedingly diseased when the cerebral and other arteries of corresponding size are almost normal. We have placed in the Museum some examples to prove this. It is likewise true that the cerebral or coronary arteries may be much diseased while the aorta is normal. The radial artery also may be sound when the cerebral arteries, &c., are bad, and the same is true *vice versâ.* We have before mentioned the occurrence of thickenings of the renal, hepatic, and other arteries, along with diseases of the viscera they supply.

The vast importance, variety, and universality, of the results of this disease make it necessary that we should have a clear view of its causes. It was long ago suggested that the true cause of "atheroma" is overstrain, and this view was held by Dittrich before the irritative earlier stage was known, and in this country the same was taught by Kirkes and others. Careful consideration of our experience convinces us that this is the true cause.

The reasons which tend to induce this belief that mechanical strain is the main cause of atheroma of the arteries may be enumerated with advantage; many of these reasons, which we think amount in all to very sufficient proof, have been urged before, but we hope to show that some facts that are often thought to oppose this belief are really in its favour.

Atheroma is more common in men than in women, and men are accustomed to more laborious work. Among men the greatest amount of atheroma is found in those who use the greatest amount of muscular exertion, *e. g.* sawyers, oarsmen, sailors, and athletes. Atheroma is uncommon in the diseases which diminish the volume of blood, and by consequence the pressure within the arteries, *e. g.* phthisis and mitral obstruction. The pulmonary system of vessels escapes the liability to atheroma almost entirely. This has been the ground for starting a theory that venous blood has some kind of opposing influence, or arterial blood has some kind of favouring influence, on the development of atheroma. But when the right heart is greatly hypertrophied from chronic bronchitis or mitral disease, the pulmonary artery then becomes affected with atheroma, although its blood is more venous than usual under these circumstances. The earliest appearances of chronic atheroma in arteries occur at points where the strain upon the coats is greatest. The obvious example of this is in the case of the convexity of the aortic arch, which is very exceptionally obnoxious to this change, as is the bifurcation of the vessel. But there are other examples of this truth in the cerebral, splenic, and cardiac, arteries.

The peculiar liability of these vessels to atheroma is generally admitted, and it is often supposed to prove that mechanical strain cannot be the determining cause of the atheroma. But if we consider the circulation of the brain, heart, and spleen, we shall find peculiarities suggesting very plainly that their circulation is carried on under special mechanical tension. Thus, the *brain*, by means of its *superior cerrebal veins*, discharges its blood forwards into the longitudinal sinus, in a direction opposed to the current in the sinus which runs backwards; this must make it difficult for the blood to escape from the brain, and thus lead to resistance to the flow from the arteries and tension within them, straining them, spoiling their nutrition by irritation, and so leading to "atheroma," and at last choking or bursting them, when the chronic impediment has lasted years enough. The state of the vessels is again mentioned under "Brain." The *heart* is very peculiar in its parietal circulation, seeing that the ventricular contraction throws blood into the *coronary arteries*, while it hardens and compresses the tissues of the heart, so as to impede at the same moment the passage of blood on through the capillaries, thus producing tension in the artery from the resisted current. This, we think, is sufficient to lead us to anticipate that the heart's vessels would, sooner than others, suffer from any weakness, such as atheroma, which pressure elicits. And it may be that the direct causation of the impediment to the arterial blood in the athlete is principally due to the opposition to the arterial flow in his muscles during their over-strong and over-long contraction. The *spleen's* circulation shows remarkable peculiarities, for great venous spaces are constructed in it, which have to empty themselves into the liver; and we know how easily the spleen swells under hepatic obstruction. It has therefore to endure an impediment to the onflow of its venous blood, and this will entail a resistance and tension in the *splenic artery*. This tension will strain the artery and lead naturally, on the view we are advocating, to atheromatous disease of its vessels, to which it is very liable. The only other organ which shows (but under prescribed conditions) much of such excessive liability to atheroma of its arteries is the *kidney*. This organ, however, only shows the affection in chronic Bright's disease, with contraction, and wasting of its cortex. Now, this disease goes with wasting away of the capillary glomeruli in the Malpighian corpuscles of the kidney. One example, in which we made the observation very carefully, showed, in a fine section of the cortex, thirty-two wasted and closed-up glomeruli to eighteen healthy ones; now, this closure of glomeruli must greatly increase the strain in the renal artery, and thus explains why, in old Bright's disease, the artery thickens, and becomes atheromatous.

The arteries of the lower extremities are more liable than those of the upper to atheroma. Now, it may be thought that, as the lower limbs are more constantly directed downwards than the upper, the strain in their arteries would be alleviated by the gravitation of their current, but it must be remembered that the current in the veins has a longer distance to achieve under difficulties to which those of the upper are strangers. Under these circumstances the greater liability of the femoral arteries than the brachial to atheroma is much in favour of the belief that mechanical tension is the cause of the atheroma.

The appearance of the interior of the aorta in the early stages of atheroma supports the evidence that the occurrence of it is due to tension within the vessel, for the atheroma is generally not distributed without a certain suggestive pattern. It observes this rule, that the atheroma forms lines lengthwise to the vessel's course, that is, across its transverse girth. Now, the longitudinal tension in the vessel's wall is not so great as the transverse, if only for this reason, that the tension wave of the pulse passes as a transient moving strain down the vessel, so that the longitudinal strain pulls on the unstrained part which the strain has left or not yet reached, and so is relieved, while the transverse strain is all round alike, and has no escapement. Now, this transverse strain will, of course, tend to tear the coats across its own direction, that is, it will tend to tear longitudinal rents; but though it does not effect this, it produces chronic longitudinal weaknesses and irritations that evince themselves as atheromatous patches.

We might carry much farther the argument, showing that those chronic blood impurities, the chemical diatheses, uric and oxalic especially, go with a tendency to thick and strained vessels, and so at last with atheroma. All this, probably, through the resistance which impure blood meets in the capillaries, and the tension in the arteries which this resistance creates, so that dyspepsia thus becomes a source of atheroma, through the medium of mechanical strain.

It seems to us that evidence enough has been given that what is called atheroma of arteries is sub-inflammation of various degrees, of which the lower degrees end in fatty degeneration of the coats, along with the inflammatory products; and that the determining cause of the occurrence of this change is mechanical strain. This by no means interferes with any belief that a general altered nutrition in gout, syphilis, &c., may lay the coats of vessels more open to suffer from the said strain, and we are disposed to think that it is probably true that they do so; but no one has yet shown this to be true.

Besides the fatty and calcareous degenerations of the arterial walls which we have just now spoken of as the results of deforming arteritis we meet with simple fatty and calcareous degenerations which are primary, and not preceded by inflammation.

*Simple fatty degeneration* appears in the form of opaque whitish spots marbling the inner surface, which at such spots is scarcely elevated, if at all. Microscopically, the change is seen to consist of a deposit of fat in the cells of the innermost layers of the inner coat. These cells are of stellate figure, and their branches intercommunicate. When healthy they are not easy to see, but when thus loaded with fat they become very conspicuous, so that the inner layer of such a vessel, when stripped off, forms a striking microscopic object. The further progress of this change leads to giving way of the fatty tissue, so that there is a very shallow breach of the surface, which is slightly downy at the affected spot, but the substance of the wall of the vessel beneath is practically unaltered. On the whole, this simple fatty degeneration is not an important disease; it is very common in the aorta; indeed, it is rarely that you meet an aorta of more than two or three years of age that does not show some of it. The change is not often seen, however, in the middle-sized arteries, especially those of the limbs. But in the smallest vessels distributed within some organs, especially the brain, a fatty degeneration, shown in the collection of fat-grains around the nuclei of the cells in the adventitia, is very common in later life. This is a more serious disease; it leads to rupture of the vessels, producing apoplexy, &c.

*Simple calcareous degeneration* is another mode of senescence in arteries; it is found chiefly in the middle coats of those of medium size, especially in the lower limbs. It affects the muscular fibres, and hence appears as bands running partly around the vessel, which at the same time is irregularly dilated through loss of its elasticity; the bands may form complete rings, and if the change progress to an extreme extent the artery may be transformed into a stony tube. Then arises a danger that coagulation within the vessel will cause senile gangrene.

Thus, as to the simple degenerations, it appears that the internal coat of the largest vessels is more liable to simple fatty degeneration. The middle coat of the medium-sized vessels is more liable to calcareous degeneration, while the outer coat of the smallest vessels is more liable to fatty degeneration.

While speaking of degenerations of the vessels we must draw attention to the curious liability of the smallest arteries of some organs, as the liver, spleen, kidney, &c., to *lardaceous disease*, so that the arteries exhibit the change before it is seen in any other components of those organs, but this liability does not extend to the larger arteries. Indeed, the small arteries escape entirely in parts whose tissue is not liable to lardaceous change, so that the disease evidently belongs to the organ affected, and the implication of the arteries so early in its course is an interesting instance, showing how the mode of nutrition of an organ governs the nutrition of the apparently independent tissues that enter to supply it. It is only the main arteries that have completely independent laws of maintenance, like the Roman *primæ viæ*. The small arteries, like the *viæ vicinales*, are under the control of the neighbourhoods they serve. Their diseases are commonly due to special faults of the organs they supply, while the aorta and its great branches have their own independent causes of disorder.

**Aneurysm.**—Although everyone, no doubt, understands what is meant by an aneurysm (a sac communicating with an artery), yet the term is somewhat difficult to define accurately; and this is not so much on account of any obscurity in the disease itself, as because authors have adopted such various names in connection with it, and sometimes have used the same terms in opposite senses. This is especially the case with reference to the terms *true* and *false*, which so puzzle students, owing to their different and even opposite applications to aneurysm. There are also various names given to aneurysms, some denoting the arrangement of the arterial coats, others the form and size of the sac. First, with respect to the terms *true* and *false*, and the manner in which they have been used. Scarpa, one of the earliest exact writers on the subject, from failing to find all the coats of the vessel in those aneurysmal pouches which form tumours and contain ante-mortem coagula, described this absence of the coats as the true condition of aneurysms; whereas he called that a dilatation, or false aneurysm, where all the coats remained, and no coagulation had occurred. Now, it is remarkable that the very opposite terms have been given by many authors to the same conditions, and aneurysms have been called true where all the coats are perfect, and false where some of the coats were wanting, such authors evidently being influenced by the connection in their own minds of true with perfect and false with imperfect. But the idea of Scarpa has a certain practical value, at least so far as it applies to aneurysms of the aorta, for no two affections can be more different than the dilatation of the whole vessel on the one hand and the formation of a pouch from its side on the other. The one case interferes with the circulation and the heart's action, while the other only produces effects of pressure or mechanical thrust on surrounding parts, like an ordinary tumour; the one consists, as Scarpa truly said, of all the coats, and contains no coagula, while the other, if of any size, is deficient in the coats, and contains coagula. Although he was so far right, it certainly was incorrect to limit the name true aneurysm to that only where the coats were deficient, for the early stage of an aneurysm is generally a bulging of the whole arterial wall at the spot affected (the exceptions being rare instances of rupture of the artery). So that, at first, all the coats of the vessel may be detected in the wall of the sac, though truly in a very altered condition, and very soon disappearing, so that we seldom find the middle coat in a sac which is bigger than a plum. The forms or shapes of aneurysms give rise to many names for them. Thus, when the whole circumference of the artery is dilated, and the enlargement circumscribed, the term *fusiform*, *cylindrical*, or *globular*, is used. Sometimes such a dilatation of a limited part of the whole trunk is abrupt, reaching suddenly a large diameter; it then may turn over the border of the opening in the parent vessel, and extend up or down it a greater or shorter distance, so that the opening of the vessel makes a projecting lip within the sac. The aneurysm is then said to be *invaginated*. The term *saccular* is applied to the commoner form of aneurysm, where a pouch comes off from one side of the vessel. Although the coats vary in different cases, the term *true* aneurysm may be used for all these. We then use the ordinary word *false* for aneurysms in which the coats are not expanded, but burst through, that is, where none of the artery is in the sac it apparently gives off. Finally, when upon rupture of the sac the blood is dispersed among the tissues without a containing boundary, the old word *diffused* cannot be improved on to signify the accident. In these cases a distinct sac may form after a very lengthened period.

Sometimes we see saccular aneurysms come off from fusiform ones, &c. Indeed, endless combinations of all these varieties may be met with in the aorta, when the arterial disease which has given rise to all these alterations of form is extensive and severe. Practically, if the sac of the aneurysm be of some size, it is formed principally by the external cellular coat, the internal coat proceeding only a short distance inwards; within this we find fibrinous laminæ, which form in the part of the sac farthest from its mouth, as concentric layers. These laminæ are of a pinkish colour, are very thin, and lie one upon another in great number, so that a section resembles that of an onion. Their surface presents a remarkable appearance from there being a series of parallel ridges upon them, resembling the "ribbed sea-sand." Their substance is tough, and of a buff colour, not unlike that of the internal arterial coat, but they are apt to soften down like other old fibrinous clots, and sometimes such clot will move on out of the aneurysm, and plug the artery beyond.

The amount of this laminated deposit increases as the blood within the sac is allowed to rest, so that in fusiform aneurysms, where the blood freely courses throughout, there is none of it. But in saccular aneurysms there is a varying quantity, the amount being large when the sac has a mouth that is small in proportion to its space; when the mouth of the sac is large and the sac small, so that the blood rushes freely in and out through it, then there is very little clot formed. The operation of ligature or pressure aims at securing an entire cessation of motion in the sac. It is said that sometimes the aneurysm bends over on to the proximal side of the artery so as to compress it and thus shut off the access of the current, and cure itself. But against the chance of this we must reflect that the very pressure closing the artery would be the same pressure as that within the artery it is supposed to close, and, in fact, instances of this occurrence are as difficult to find as this reflection would suggest.

As aneurysms of the aorta are the most important, we will look at some of the best-marked specimens. In one we see a small pouch between the aortic valves, and projecting upwards between the auricle and ventricle; such aneurysms, we think, are produced by endocarditis where the valves are involved, and are similar to those we have already described under the term acute aneurysm of the heart, for we can scarcely call a pouch between and under the valves an aneurysm of the aorta. If next we follow the course of the aorta, the first aneurysms which occur are immediately above the valves, in the sinuses of Valsalva; these are sacculated, have small openings, and generally give way before they have reached any great dimensions. Thus in one case, where such an aneurysm opened into the right ventricle just below the sigmoid valves, the patient's life was protracted for a considerable time. We might mention that these aneurysms at the commencement of the aorta may interfere with the closure of the valves, and consequently the imperfect action of the latter becomes the main disorder. In another specimen an aneurysm is seen protruding into the right ventricle and pulmonary artery, and sometimes such an one bursts into the artery, or such aneurysms may burst into the left auricle or the right auricle or the right ventricle. More usually these pouches at the beginning of the ascending aorta rupture into the pericardium, and instant death is the consequence. On opening the pericardium in such cases we first of all see only the serum, which has separated; but on removing this the heart is found surrounded by a black coagulum of blood, which has come through a hole at the root of the aorta. The loss of blood is not much, and, therefore, the body is not blanched, as is so often the case from ruptured aneurysm elsewhere; for, in this case, death arises from mechanical interference with the heart's action, and the blood is prevented flowing from distant parts, and therefore we meet the apparent anomaly that death has been caused by a ruptured blood-vessel, and yet the tissues are found congested. In like manner distinct pouches form higher up, until we reach the lower part of the arch, where the vessel meets the spine, a very favourite seat for the disease. Aneurysms take different directions according as they are situated in the ascending or descending part, and, consequently, press upon or rupture into various parts, as the œsophagus, or the trachea, or bronchi. One very important effect produced by aneurysms in this part is due to the pressure they exert on the pneumogastric and recurrent laryngeal nerves, and in the latter case especially paralysis of the muscles of the larynx takes place, whereby stridulous breathing and other characteristic laryngeal symptoms are produced. In one specimen it will be seen how the nerve was compressed; the symptoms were wholly laryngeal, and suggested tracheotomy, though it was not performed, as the nature of the case was suspected. In another specimen, the operation took place. In some instances, where a large aneurysmal pouch forms in the ascending aorta, it bulges forward and makes its way through the ribs and sternum, but very seldom bursts externally. This is the least painful form, for it is often unattended by any pressure on important neighbouring organs; and in a case we lately saw in the post-mortem room the aneurysm was so large that it not only penetrated the walls of the chest in front, but eroded the spine behind. General dilatation of the arch, or fusiform aneurysm, may also be seen in our specimens. After leaving the arch, a very favourite seat for aneurysm is where the vessel meets the spine, and is in contact with the fourth, fifth, or sixth, dorsal vertebra. The aneurysm may form on the front, side, or back, of the vessel; though, when large, very much the same effects are produced, as the enlargement takes place backwards and laterally. One of the most marked effects seen as the result of aneurysm at this spot is an eroding of the dorsal vertebræ, so that when the vessel is removed their bodies may be seen quite eaten away, leaving the cartilages projecting and unaffected. In some cases the destruction of the bones may go on to the extent of laying open the spinal canal, so that, as in one case, the finger could be passed from the aneurysm into the canal, and the patient, as may be supposed, had paraplegic symptoms. In such cases the wall of the aneurysm has long disappeared, so that the blood within, or its coagula, are in actual contact with the spine, the wall of the aneurysm merely closing in the diseased spot on each side. In this very common form the sac is generally oval, and communicates with the artery by a long oval opening. If the aneurysm should come off from the side, it soon reaches the spine, when the same effects are produced, only then the absorption is more lateral, and the ribs may be involved. Even if the aneurysm be formed on the anterior part of the aorta (which is not often), it may bulge over on one side, and affect the spine in the same way. Such aneurysms may cause death, as before mentioned, by opening into the trachea, bronchi, or œsophagus; or they not uncommonly burst into the chest, producing sudden death; in some instances they penetrate the lung, as we lately saw, and, making their way through this organ, reach the parietes of the thorax. They also sometimes extend beneath the costal pleura, which thus gives them a false covering. If the parts near which they run are not actually laid open, we generally find these altered in form by the pressure, and thus we meet with a flattening of the œsophagus or bronchi, and very often disease in the lung; the immediate cause of death being pneumonia. Aneurysm is more rarely met with at the lower part of the thoracic aorta, and may be of the fusiform or sacculated kind. But we have a specimen of an aneurysm opposite the bifurcation of trachea, and another at the diaphragm. After leaving the arch of the aorta, the next most frequent place for aneurysm is where the vessel passes through the diaphragm, so that sometimes a part of the tumour is seen in the chest and a part in the abdomen; or perhaps it is more frequent a little below this spot, and near where the cœliac axis comes off; the effects differ according as it is formed from the posterior or anterior part of the vessel. Seventy per cent. of abdominal aneurysms are at this spot. If from the back, it absorbs the vertebræ, as before seen; but if formed in front, a tumour projects anteriorly, which involves in it very often the branches of the cœliac axis; these come off from the aneurysm, or pass along its sides, and are compressed by it. In these cases the semilunar ganglia and cœliac plexus are often involved, and probably produce some of the symptoms. More rarely aneurysms occur in the course of the branches of the abdominal aorta. Thus we have an example of aneurysm of a branch of the colica sinistra, with another in the same case on the gastric, and two on the cystic arteries. Such aneurysms of small arteries are sometimes evidently due to embolism. Another is an aneurysm on an arterial twig in the forearm, and one on a meningeal twig in the dura mater in the same case. These examples are from two cases of ulcerative endocarditis, and, no doubt, were due to embolism. In one case also the femoral artery of a boy with ulcerative endocarditis shows a large aneurysm also due to embolism. In cases of aneurysm in young persons there has often been a history of acute rheumatism. It was so in a case of axillary aneurysm in a girl; also in a case of aneurysm of the mesenteric artery. In these rheumatic cases there is always a probability that rheumatic carditis and embolism may have been the cause of the aneurysm; but, we may ask, does it happen that along with rheumatic endocarditis there may also be a rheumatic endarteritis, weakening the affected spot and leading to dilatation and aneurysm? We have occasionally met with a patch of inflammatory softening of the aorta near, but not continuous with, aortic valves that were acutely inflamed from rheumatic fever, but in all instances but one there was reason to ascribe the aortitis to impact of vegetations attached to the valves. So that we know of but slight grounds for supposing that there is an arteritis directly rheumatic.

From the number of instances now recorded there can be no doubt of the origin of aneurysm in embolus. In nearly all the cases where young people have been the subject of aneurysm of the smaller arteries an embolus has been found in connection with it, and occasionally the aneurysm is seen in the process of formation. There may be other special conditions required for the aneurysm to occur, and Dr. Goodhart suggests that the embolus sets up a low septic inflammation, which causes a softening and bulging of the coats.

In the middle-sized arteries of the limbs aneurysms are very common, and our shelves show the several varieties, and the effects of tying the vessel above them, and the large anastomotic vessels resulting. We have a remarkable specimen where an aneurysm of the iliac artery burst into the cæcum, causing death by hæmorrhage from the bowel.

Aneurysms within the cranium are frequent as compared with their rarity elsewhere in vessels of the same size; we might, perhaps, expect this, remembering how thin are the intra-cranial arteries. They often occur in young persons, a large proportion under the age of twenty-one; and they are then mostly, but not exclusively, embolic. If small, they may give rise to no symptoms until they burst, when the patient is suddenly seized with sanguineous apoplexy; if they be in the cerebral substance, softening may ensue; or they may produce local paralysis. We have met with a case of aneurysm within the cavernous sinus which paralysed the motor nerves of the eye and then burst into the middle lobe of the brain, as in one of these cases where the tumour pressed on the third nerve, producing paralysis of ocular muscles. Aneurysms are occasionally met with in the coronary vessels of the heart, and there is a specimen in St. Thomas's where a number of small aneurysms exist upon them, giving them a beaded or varicose appearance.

Very small aneurysms are called miliary aneurysms. They are mostly met with in the cerebral vessels, but have been found occasionally in the kidney and mucous membrane of the alimentary canal. The aneurysms are mostly fusiform, and do not show any disease of the arterial coats.

There is every reason to believe that an aneurysm may be due occasionally to syphilis.

*A false aneurysm* is a sac filled with blood communicating with an artery, but the walls of the sac not formed from the vessel itself. It is most commonly *traumatic*, being produced when the artery has been punctured; as, for example, the femoral, by a knife or sharp-pointed instrument, when the blood escapes into the tissues around, forming a circumscribed pulsating tumour. This is a form of disease which will be fully considered in the surgical lectures, as there are very important points of treatment connected with it.

*Aneurysmal varix* is where the artery has been punctured through a vein, so that the two communicate, a subject also fully treated of in the surgical lectures. We have a specimen showing a communication between the abdominal aorta and vena cava which produced dropsy. It probably originated in a small aneurysm.

*Varicose* or *general dilatation of the arteries* is occasionally met with; a most remarkable case is reported by Mr. J. Adams, of the London Hospital, in which the patient had all his arteries, or at least those of his limbs, immensely dilated, being two or three times their natural size; and the consequence was a fatal hæmorrhage from an ulcer.

*Dissecting aneurysm* arises when blood bursts through the inner coats of a blood-vessel, and passes down between its outer coats; it has generally been thought that this occurs between the external and middle coat, but Dr. Peacock has stated that the middle coat itself is sometimes split, so that one part of the middle coat is united to the intima and the other to the adventitia. The affection arises from the more chronic form of arteritis, as before stated, the blood entering an atheromatous sac or passing behind a calcareous or otherwise affected part of the coat.

Sometimes we see remains of a former attack, in the presence of whitish fibrinous layers between the coats. In some cases the blood penetrates the internal diseased coat, and, after passing down a greater or less length of the vessel, re-enters by bursting again inwards through the coats. Under these circumstances recovery may ensue. A most remarkable example of this is given by Dr. Fagge in the 'Medico-Chirurgical Transactions.' The proper coats of the arch of an old gentleman's aorta were torn through, and the blood passed down between the middle and outer coat on one side of the aorta through its whole length, reaching to the common iliac arteries, into which it again burst by a distinct opening on each side. In this remarkable case the patient lived some years after the rupture of the vessel, and the new channel took on so much the appearance of the interior of an artery that it was quite difficult to say which was the proper aorta and which the adventitious, for the latter had a perfect lining resembling exactly the inner coat of the artery, and having, like this, patches of fatty degeneration resembling ordinary atheroma. Dr. Peacock has published cases where some time elapsed before the rupture of the external coat; in the meantime a distinct pouch had formed between the coats of the artery, and this was lined by a smooth membrane. Occasionally after rupture the internal coat may heal, and fissures may be found with smooth edges united by fibrous bands.

Sometimes an aneurysm treats the œsophagus in the same way in separating its coats. Thus, we have seen an aortic aneurysm burst in between the muscular and mucous coat high up the œsophagus, and run down between these coats to burst into the stomach at its middle.

**Aneurysm by Anastomosis—Telangiectasis—Nævus.**—Growths composed principally of blood-vessels, or of tissue communicating with the blood-vessels, may affect various parts, but especially the skin. They are for the most part congenital, though they may increase rapidly after birth, and occasionally, we think, they may commence at any period of life, especially after an injury. They have generally been divided into three classes, according as the capillary, venous, or arterial element prevails; and no doubt many varieties exist in this respect. Thus, sometimes a vascular growth may be seen to be full of venous blood occupying sinuses; at another, large arteries lead into it, by which a pulsation is imparted to it; and in other cases, as so often seen in nævi of young children, the vascularity is due to an increase of the smallest vessels, or capillaries. Besides these, many nævi consist of a new tissue, composed of cellular or membranous structure, forming spaces in which the blood is contained, this cavernous tissue communicating with the blood-vessels; whether this tissue is formed from the vessels, as the veins, or is a distinct formation which subsequently becomes vascular, we think is scarcely yet made out. A remarkable case of pulsating tumour of the foot was examined by Mr. Howse, and found to consist of a spongy substance indirectly communicating with the arteries and veins. It seemed as if it grew out of the veins, although it could be injected from the arteries; at all events the trabeculæ directly joined the vein walls. We shall, when coming to Liver, be able to show some specimens of cavernous tissue in that organ communicating with the veins. A change which nævi sometimes undergo is called the cystic degeneration; thus, after their existence for a certain time, a number of cysts form, and the vascularity is in great measure lost; it is indeed often a question for the surgeon, whether some of the cystic tumours of long standing which he removes have originally been nævi or not. Thus in a specimen, consisting of a mass of cysts now empty, which Mr. Cock removed from a boy's neck, there were large blood-vessels passing into it, and filling the spaces with blood, so that it nearly emptied itself, after removal. The growth appeared intimately connected with the blood-vessels of the part, but there was no unequivocal history of its having been preceded by a nævus. A nævus on the skin is a mother's mark; when covered with hair, a hairy nævus; and when it involves the subcutaneous tissue, it constitutes a tumour.

**Injuries.**—*Rupture of Aorta.*—We have mentioned the occurrence of rupture of the vessels through disease of their coats; but there is also a small class of cases in which the aorta suffers rupture, generally by reason of hypertrophy of the heart. The laceration is mostly transverse, but it has been seen to have a longitudinal direction; it usually occurs in the arch of the aorta. This accident will of course cause sudden death, but if the rupture spare the outer coat a dissecting aneurysm may result, as in the remarkable case of Dr. Fagge's, just related.

Injuries to blood-vessels form a subject more proper to the surgical lectures, but we should notice carefully the liability of the great arteries to be torn through in fractures and dislocations, a very serious complication of such accidents; or the abdominal vessels may be burst in crushing injuries to the abdomen, it may be the renal or the mesenteric. Sometimes the aorta is pierced by instruments or lacerated by the rough contact of a fractured sternum. Such injuries are usually immediately fatal, but a small puncture may kill more slowly. Thus, a needle wound of the ascending aorta caused death in two hours. Sometimes the aorta is lacerated through injury in such a manner as will prove it to have been burst rather than torn by rough contact. In fracture of the sternum we have found a rupture of the aorta in the concavity of its arch, while the convexity which is towards the broken bone was not injured.

A very important example of injury to great blood-vessels, to which we would draw attention, is met with in some cases of tracheotomy. The end of the tube causes ulceration of the trachea, the ulcer spreads through its wall until the tube may reach the innominate or common carotid artery, producing immediately fatal hæmorrhage.

**Morbid Growths.**—*Tubercle* is not known to attack blood-vessels, but miliary tubercle may grow on the blood-vessels, as seen in the brain and heart. Lancereaux describes small gelatinous tumours sometimes found on the blood-vessels of the brain, and occasionally small cysts which have sprung from the lymph spaces. *Cancer* never primarily affects the arteries. They are also very callous to the encroachments of cancer when reaching their walls from other parts. It is remarkable how long a time a great artery will resist all the operations of a growth of cancer outside it, and thus we often find a large mass of cancer, as, for example, in the mediastinum or the lumbar glands, with the aorta passing through its midst quite unaffected, and usually without compression or other alteration of its calibre. The veins, on the other hand, are generally implicated in the growth. Rarely, however, and in exceptional cases, the coats of the arteries may become involved, especially the thinner coats of the pulmonary artery in mediastinal cancer, but even then we have never seen the elastic coats changing to cancer like other tissues, but they are pressed in and wasted away by the cancer, and so a cancerous nodule may be seen projecting into the interior. When, however, a large artery is near a cancerous sore in a sloughing state, the vessel's wall is apt to give way along with other tissues in the sloughing process. This generally results from sloughing epithelial cancers. Thus such cancers in the pharynx or œsophagus may reach the carotid, or cancer of the œsophagus only may reach the aorta.

# DISEASES OF THE VEINS

**Malformation.**—The various deviations from the normal standard of distribution of the veins will be pointed out in the anatomical lectures. We have a heart where will be seen the left innominate vein passing down by itself at the back of the organ to enter the right auricle separately, instead of crossing in front to meet with its fellow, and another curious case, where the right pulmonary vein gives off a long channel, which runs straight forward and ends in a cyst of the size of a plum, which lay adherent on the inner side of the front of the lung between it and the pericardium. It was full of a clot which was evidently of post-mortem formation.

In connection with this we will mention certain well-known curious blood-cysts that occur about the neck as well as in other parts. Their origin is obscure, but some have been traced into continuity with large veins (*e. g.* the femoral). A very large one, which was removed and has been described by Mr. Birkett, extended from the neck into the axilla. Some of these cysts have been clearly traced to the dilatation of the vessels in nævi. Others arise by hæmorrhage into simple cysts. These are thin-walled cysts with fluid blood in them, and must not be confused with those blood-clot tumours in the limbs which are so apt to prove malignant. The latter generally show clot of various date, and careful search will generally discover a patch of malignant growth in which the vessels have given way and caused the hæmorrhage.

**Inflammation of Veins; Phlebitis.**—In considering inflammation of the arteries we found that acute inflammation of them is very rare, and that when reached by neighbouring inflammation they show very little disposition to adopt its activity, being, indeed, of remarkably callous nature, more resembling cartilage in this than any other tissue. Now, this is not quite the case with the veins, for although phlebitis once was thought to be more widely spread and important than it really is, this must not induce us to overlook the fact that acute inflammation of veins is a tolerably frequent and often a very serious occurrence.

When a vein is acutely inflamed it is always found full of clot undergoing changes. The amount of clot distends the vessel so as to make it feel both during life and after death like a hard cord. At the same time the wall of the vein is thick, and when cut into it appears as substantial as the wall of the artery. If the inflammation be very severe we find pus between the coats, perhaps in several layers.

Now, there can be no doubt that in many cases the clot found in the thick-walled and inflamed vein is truly blood-clot. But in other cases there must be equally no doubt that, along with the blood, inflammatory products are present until the clot may have all the microscopical characters of pus.

At the risk of being tedious we must shortly state the present position of the question of phlebitis, which is always one of the most important questions in pathology, and, in some form or other, has exercised great influence on the views of pathologists at every period.

It will, we think, be best for us to state the intricacies of the subject with as little entanglement as possible. Puriform matter had long been known to occur in the veins, and was accepted as pus. Starting then from that time when puriform matter in the clots within inflamed veins was accepted as purulent, which was the first and natural view taken of it, we first meet with a denial of any such purulent nature of clots within veins by Goodsir and his followers. This denial was based on the microscopic appearance of the puriform matter, which was stated to be that of molecular detritus and not of pus. This denial had become universally prevalent, but in the last few years it is very gradually being admitted that the microscopic evidence of the non-purulent nature of this puriform matter is not so decisive as was until recently assumed, so that explanations are wanted. Virchow, for instance, and others, allow that a large number of pus-like corpuscles are present in the puriform matter, but they explain these pus-like corpuscles by pointing out that white blood-cells are less destructible than red ones, so that when the red corpuscles in a clot have dissolved and been washed out of the clot, the white corpuscles still remain and appear as pus-cells. But then sometimes there are far too many pus-cells to be explained in this manner; hereupon Rindfleisch and others appeal to the adhesive property of the white blood-cells, which induces them to cling to the surface of the clot or to penetrate within its spongy texture, fresh layers of clot forming then over the layer of such adherent white cells; when such a complex clot is supposed to soften, then it is thought that a sufficient explanation of the large number of pus-like cells is achieved. But we must remind ourselves here that the evidence has undergone a curious revolution from Goodsir, who bases the non-purulent nature of clots on absence of pus-cells, to Rindfleisch and others, who explain away their presence.

In the meantime, however, the well-known experiment of Cohnheim shows that the pus in the inflaming mesentery or cornea of a frog is nothing else than out-wandered white blood-cells; thus, white cells out of place claim to be pus-cells. Here, then, we find another awkward complication thrust on the question, especially since Weber has caused the white cells of the blood to take up cinnabar after a clot has first been formed, and then proved that these cinnabar-bearing cells find their way into the clot after its formation; such white cells wandering into the clot are as much pus as the white cells are when wandering out of the vessels in the mesentery. Thus, on the view of suppuration, which is based on Cohnheim's experiments, it is difficult to distinguish the pus-like cell-bearing matter in such clots from true pus; in fact, on that view it is pus.

We go over all this rather intricate ground because it is very necessary to be clear about the nature of the processes within the vessels; for, although the details of interpretation may not be altogether agreed on in the most modern times, yet it has been certainly shown that the formation of these clots and their effects on the vessel they form in, and the effects of their removal to remote parts, play a supremely important part in many grave and fatal pathological processes.

We shall endeavour to show that some clots contain pus and some do not, and for this purpose we will leave the theoretical consideration of the matter and consider the different anatomical forms in which phlebitis is met. As in other inflammations these are partly different degrees of severity, partly different kinds due to unknown differences in their causes.

*Pus in the veins; suppurative phlebitis.*—The channel of a vein sometimes forms part of an abscess; thus, an abscess of the liver bursts into an hepatic vein and the pus passes up the vein some distance, being shut off by a fibrinous layer from mixture with the blood beyond; or the pus may pass up along the vein further and reach the cava and go up beyond and enter the right auricle of the heart; or an abscess about the hip will so open the femoral vein that a part of its channel extending up into the iliac vein shall be like an open offset from the cavity of the abscess. The presence of pus within the vein is in such cases indisputable, the wall of the vein is found to be altered by thickening and formation of granulations on the surface, or by pus in layers in its substance. But we have found the veins near suppurative inflammations containing true pus when there was no continuity of their interior with an abscess. This is, indeed, frequently the case with the veins of an ill-conditioned stump, after death by pyæmia following amputation or acute necrosis, compound fracture or burn. We have seen also in such cases a clot, containing what microscopically was pus, lodged in the pocket of a valve in the femoral vein at a distance above another purulent clot. In a case of pyæmia from a bubo there was a pus-clot which went up the iliac vein from the femoral and then extended down the inferior hæmorrhoidal veins to an inflamed pile. This clot was full of pus and there were antemortem polypi in the right auricle also showing pus.

Another grave and important kind of case is where there is purulent clot in the sinuses of the dura mater; this is generally set up by caries of the neighbouring bones, especially the bones of the ear, or else it may be caused by traumatic ostitis with or without fracture, but it is not exclusively due to disease or injury of the bones, and may arise through extension of phlebitis along the facial veins, a terrible risk incurred in cases of malignant pustule, as we have several times verified, but occasionally met with in cases of ulceration in the nasal fossæ, &c. Again, in operations about the uterus or bladder the pelvic veins may show truly purulent clot extending up their ramifications as far as the iliac veins. This is the best established cause of phlegmasia dolens in lying-in women. In all these cases the inflammation extends from the minuter radicles of the veins up the branches to the trunk; but sometimes a vein suppurates through injury, as after phlebotomy, or in the interesting class of cases of inflammation of the umbilical vein in new-born infants, in which suppuration may extend into the interior of the body, producing abscess of the liver or peritonitis.

The means of conveying the inflammation in all these examples is the clot formed in the blood, which clot extends up from twig to branch, and from branch to trunk. This clot, poisoned with the diseased juices from the wound, and purulent with in-wandered white blood-cells, inflames the wall as it spreads. We cannot join with those observers who treat of the clot as if it were always simple fibrin undergoing only ordinary changes of simple decomposition. These observers do not give weight to the truly purulent microscopic character of the clot, following Goodsir's account of thrombus which Virchow adopted so implicitly; but Goodsir's account of soft thrombus, as only detritus, applies only to thrombus within the heart and non-inflammatory or sub-inflammatory thrombus generally; while we are now bound to admit also an inflammatory thrombus which contains pus. Whether this pus truly arises by wandering in of white cells out of the blood as in pus-formation outside the vessels by the wandering out of these cells, or whether it be due to some process of pus-formation not yet cleared up, is a question in the explanation of the presence of pus; but there can be no question as to the fact of its presence in the cases we are speaking of, and we can no longer regard the pus-like matter as a change in the clot. Thus, Dusch, in his admirable 'Monograph on Thrombosis of the Cerebral Sinuses,' assumes that all puriform products described as pus by their actual observers were mere softened thrombi, although he draws an important distinction between clots caused by mechanical stasis, and clots due to the neighbourhood of suppurative inflammations extending to the walls of the veins.

*Primary suppurative phlebitis* is, fortunately, very rare; so rare is it that it is constantly ignored. It is, perhaps, most certainly recognisable in the portal vein. Many cases of suppurative *pylephlebitis* have been recorded; we ourselves have met with examples. Under Liver we shall speak of this again. We have met with suppurative pelvic phlebitis in gonorrhœa without abrasion of the mucous membrane, and a similar case occurred some years ago under Dr. Habershon's care. Both these cases ended in fatal pyæmia.

*Adhesive inflammation of veins, and chronic or laminated thrombosis.*

—Plastic inflammation or "sub-inflammation" of the veins is of frequent occurrence. We occasionally find a vein, which has been pressed flat by a neighbouring tumour, closed up by adhesion of its apposed surfaces, &c. But plastic phlebitis is generally a consequence of thrombosis in states of low pyrexia. (*Thrombosis* is a coagulation of the blood *in situ.* A *thrombus* is a clot formed *in situ.*) We will now recapitulate the causes of coagulation of blood in the vessels. They are—Stoppage or extreme slowness of the current. This is one effect of roughness of the containing surface as the blood is retarded in its course. Contact with an abnormal surface, such as a surface deficient in vitality or altered in its vitality. Increase of fibrin in the blood, either an absolute increase or an increase of the coagulating tendency of the fibrin, such as is observed especially in low febrile states and cachectic inflammations, in pregnancy, &c., or the existence of some other fibro-genetic substance favouring coagulation. Much attention has of late been given to the presence of the so-called blood lamellæ in the causation of thrombosis. When coagulation occurs in a vessel, these plates are found, but whether they form a real constituent of the blood or are merely produced from precipitated globulin has not been determined. Dr. Wooldridge, as before mentioned, doubts their importance in causing coagulation. By a little consideration we shall perceive that in bedridden persons near death, or in those excessively reduced by fever, &c., the first two conditions are likely to arise in some portions of the venous system earlier than in others, so that the blood will tend to coagulate in some parts while it circulates in others. We will shortly mention the chief seats of such occurrence. The weight of the body falls especially on the buttocks, and the gluteal circumflex and other veins of that part are much impeded, so that the stream in these branches may stay and the blood coagulate, the clot extending from small branches into larger until it protrudes into the main femoral or iliac veins, where it is increased in size by layers of clot, until it may quite stop up the femoral vein. While the clot is in the branch in which it arose it is called an *autochthonous* thrombus; where it extends into the main trunk it is said to be a *produced* thrombus. When there, it is exposed to the whole current from the lower limb, and it is liable to be broken off and carried on in the current until it is cast into the pulmonary artery as an *embolus;* or before it is so carried away, the blood in the femoral and iliac veins for several inches may be coagulated and added to it, forming a long mass which has a bluntly-pointed end towards the heart, and bears on its sides moulds of the valves it lay against. We have known such a clot extend all the way up the *vena cava,* and present its blunt point within the right auricle. If such a large clot moves up into the pulmonary artery, the result is speedy or even immediate death. Often the clot commences in the pocket of a valve. In examining such a clot we find the older portions partly decolorised and washed clean of its dissolved red corpuscles, while the newer part is mingled brownish or purplish red, according to its age. If it have lasted long in its place, it may be found softened down to a fluid of creamy consistence and having a dirty brown or purple colour often compared to that of winelees. This softened pulp in non-inflammatory cases shows microscopically little more than granular detritus, but, as we have already said, in grave inflammatory cases true pus may be found, and pyæmia coexist.

If the clot, instead of moving to embolise the pulmonary artery, stay in its place, it may disintegrate and pass away, or it may go through various changes of a developmental kind, whose end is to reopen the channel of the vein. It is said that the exterior of the clot may harden and unite with the thickened wall of the vessel and the centre of the clot soften and open up as a channel, but this must be extremely rare. Or, again, after a long period during which the clot has adhered firmly to the wall of the vessel, both the clot and the vessel may dwindle away to a small fibrous cord. Thus the vena cava may become obliterated through disease in its neighbourhood in which it becomes involved, so that at last it is found contracted or quite closed. In such a case the superficial veins become immensely distended. We have in the Museum a remarkable case of obstruction of the vena cava which came from a female patient of Dr. Rees. The interior of the right auricle was much diseased, being fibrous, puckered, and hardened; the orifice of the superior cava was quite closed, and that of the inferior vena cava nearly closed. The brachio-cephalic vein, with the jugular and subclavian, were changed to tough, rigid, solid cords.

Another part of the venous system which shows great liability to stagnation and coagulation is the pelvic plexus of veins. The functions of the generative organs require a variable state of circulation, and this tends to induce a widening of their venous system beyond what is necessary in ordinary states. Under obstructive conditions this renders stagnation and coagulation frequent. Thus, clotting may occur in the vena dorsalis penis, or prostatic plexus; coagulation from this source also may extend up into the iliac veins, but it does so more rarely than that from the circumflex veins. The clots in these veins not infrequently persist and undergo calcareous transformation, forming *phleboliths*.

Again, the sinuses of the cranium, in cases of marasmus, form another favourite seat of coagulation. The delay is here occasioned by the wasting of the brain and consequent tendency to detain blood to fill the vacuum that would arise in the closed skull. We have occasionally seen thrombus of the cerebral sinuses in children with marasmus from this cause. Dusch has drawn attention to these cases, and points out the distinction between them and thrombus from diseased bone in the position of the clots, which are symmetrical in the simple cases due to stagnation, but are found close in the neighbourhood of the disease when secondary to local inflammation. Thrombosis also occurs spontaneously in the sinuses in very anæmic women.* In these cases, as in those of children, a secondary effusion of blood or congestion is produced in the brain substance. Another not infrequent position for thrombus is in unnaturally dilated veins, such as saphenous varices or hæmorrhoids, and another very important and not infrequent position is in the portal vein in severe cirrhosis of the liver. Again, thrombus is far from rare in the renal veins. We have met with it in infants who have died of diarrhœa and wasting; in the large white kidney of Bright's disease, especially when lardaceous; and in cases of severe injuries to the lumbar region. We have once seen in a case of Bright's disease with granular kidneys general phlebitic thrombosis in the veins of the limbs.

Now, what we wish especially to enforce is the variety of results which must arise when to the simple stagnation and low vitality there is added an inflammatory febrile or cachectic state of the blood; certainly there will then be from this cause a greater liability to the coagulation through the accompanying increase of the fibrin in the blood; and thus it is that we most frequently find the thrombi in these patients. For example, in phthisis these clots are so very frequent that it is quite the general rule to find them, unless the patient is carried off by some intercurrent affection; also, it is thus that in heart-disease or in Bright's disease the clots form usually most in those with whom a pneumonia, pleurisy, or peritonitis has complicated the later stages of the disorder. In some cases this last cause of thrombosis, the altered state of the blood, appears to be the more obvious and primary cause of the change, though the situations of its occurrence still are the same as we have mentioned; so that, evidently, stagnation has its share. Thus, after fevers, especially enteric, but occasionally too after typhus or relapsing fevers, we meet with thrombus in the femoral vessels, also we have met twice with a primary or idiopathic thrombosis in that situation.

But the point of main interest is the variation of local effect of the thrombus according to the character of the general disease present. When there is a generally inflammatory or impure febrile state of the system, then the vein wall becomes inflamed, and the inflammation is more obvious and its results more grave in proportion as the general fever runs high. For instance, the clot formed by the blood poisoned from the uterine sore after parturition, or by the disordered blood of fever, produces a clot in the usual situation of stagnation in the femoral vein which behaves very differently from the clot that forms in the last stages of a cancer or other simply wasting disease. Where the clot in the febrile case rests against the vein, its wall inflames. Why this inflammation? Some set it down to simple changes in the thrombus due to decomposition; but the clot in cancer may often be proved to be much older, and yet such simple thrombus as you find in cancer cases will sometimes remain long in the veins without even becoming adherent to their walls; so that the inflammation is not the effect of duration and its consequent changes, but rather depends on the nature of the thrombus due to the quality of the blood that forms it. The blood is impure; where it circulates freely its impurity is always being partly removed by the depurating organs, but where clotted the clot becomes a reservoir for the impurities in which they can accummulate. The clot is unrelieved by the excretory changes which the blood would undergo in passing through the lung, liver, &c., and hence becomes poisonous, and irritates and inflames the vein, thus totally obstructing it by the swelling of its wall, and, perhaps, producing inflammation around, which reaches and obstructs the lymphatics, so causing more or less of *phlegmasia alba dolens*, which is the name given when the obstruction in these febrile and inflammatory cases reaches its height. In many cases the blood is undoubtedly septic, and contains bacteria. On the other hand, when there is no more than simple stagnation, the blood not being morbid, the clot will remain in the vein, and there will be scarcely any inflammatory result, so that the thrombus may not produce any phlebitis or even adhere to the wall of the vessel. Such instances are frequent enough, in cases of dropsy which die without inflammation, in cancer, &c.

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* See several cases described in 'Lectures on Diseases of the Nervous System.'

An intermediate class is found in some cases of phthisis, &c., where a simple inflammatory pyrexia coexists with the thrombosis. In these cases some slight inflammation of the vessel and adhesion of the clot is usually met with.

We have seen thrombosis of the veins of the upper limbs in rheumatic heart disease, but have not as yet been able to explain why the coagulation occurs there instead of, as usual, in the lower limbs.

Plastic phlebitis is occasionally met with in a primary form; we have seen it, especially in elderly people, with varicose veins. This state is sometimes accompanied by saccharine urine, and occurs also in gout. The superficial veins of the forearm have thus inflamed without evident cause, also the veins of the legs, especially if varicose. We have known such simple primary phlebitis produce fatal embolism.

We would draw attention to the curious absence of dropsy in some cases of extreme emaciation when the whole venous system of the lower extremities, *e. g.* one or both common iliac veins, are quite full of thrombus, as far as we could make out, entirely adherent all around the vein. Although syphilitic disease of the veins has not been demonstrated, Mr. Hutchinson believes it to exist from the fact of cases of phlebitis being cured by specific remedies.

**Embolism.**—It will be convenient now to consider the effects of the movement of clots from the site of their original formation, and their conveyance onwards by the blood stream until they reach new lodgment in arteries too small to allow them to pass. A clot which is so moved is called an embolus, and the process of its impaction in the artery is called embolism.

Being fresh from the consideration of the various kinds of clot, or thrombus, found in veins, we shall be now able to inquire what are the effects of embolism of the arteries with these several kinds of clots. First we will consider the effects of the mere stoppage of the current by the mechanical effect of the embolus. Then we will follow out the results that supervene on account of the irritative qualities of the clot.

The mechanical effects of the embolus are not quite what we should at first expect. One naturally thinks that if the artery of a part is stopped up the blood supply will be shut off, and the part become bloodless. But, when a part of an organ is embolised, the effect is that its vessels become congested—that is, if time be allowed; for in some cases, as when the pulmonary trunk is plugged, death occurs at once through the anæmia beyond; and in embolism of the femoral the limb may in like manner die and become gangrenous. To understand the congestion that arises from more limited embolism we must remember that the vessels are not passive tubes, but that they keep their size by reason of their vital contractile power, and that they dilate when that power is lost or diminished. Next we must remember that the whole blood system is in a state of tension, so that when a vessel is divided the blood gushes out, or if a tube is put into the vessel the blood runs into the tube and fills it to a certain height. Recalling these facts, we are ready to see that where an artery is plugged, the part beyond the plug, being deprived of nourishment, will lose its contractile power and give way, offering no resistance to dilatation; and meanwhile the tension of the whole blood forces some into this dilated space, as it would into a glass tube or other line of escapement—in short, there is a sort of hæmorrhagic escapement of blood into the starved, paralysed, and dilated, vessels. But, we may ask, why is it that when the blood so enters anew into the vessel, this does not recover itself and contract? Here, again, it must be observed that the blood that thus has entered has no longer got the force of the heart playing on it directly, so that it does not circulate, but is stagnant, impure, and unable to keep up nourishment. Thus there is no renewal of life except in the outer area of the implicated spot where the anastomotic vessels keep up some flow. We shall find that the results we are now describing occur by far most markedly in organs whose arteries do not anastomose, such parts as the spleen, kidney, retina, &c., while in parts where anastomosis is free these mechanical results of embolism are scarcely seen, or not at all. The stagnation of the blood sufficiently explains its inability to maintain the contractility of the vessel. But it is not only the wall of the vessel that suffers: there is a general lowering of the nutrition of the part; the over-gorged dilated vessels allow the escape of serum, and by bursting or by transudation allow blood-corpuscles to escape, and thus arises œdema or apoplexy of the part. All this has been well traced out by experimental embolism in the brain, and it has been watched with the ophthalmoscope in embolism of the *arteria centralis retinæ*, although it may not be peculiar to embolism.

In the course of a short time degenerative changes supervene. The blood coagulates in the dilated vessels, and the tissue withers. The colouring matter is dissolved out of the red corpuscles, and fatty and granular degeneration overtakes all the cellular elements of the affected tissue until the central part of the embolic patch is pale from the first and opaque from the second of these causes, often having a yellowish or brownish tinge from the presence of hæmatoidin, and being so shrunken as to appear like a scar, the fibrous parts alone remaining. But, meantime, around the seat of these changes a congestion is maintained where the force of the surrounding circulation keeps up some pressure, and also some flow, in the dilated vessels.

Such is the general mechanical effect of embolism of small arteries in tissues whose vessels communicate but little, and therefore it is in the spleen and kidney, whose vessels do not communicate much, that these results are best seen. In the brain there are special effects due to its consistence or other conditions. Again, in the lung embolism of small branches of the pulmonary artery with non-inflammatory clots, as in phthisis, and in the liver embolism of small branches of the portal vein or hepatic artery, does not produce the results we have described, because in either case the tissue has a second source of nutritive supply.

These results are due to simple mechanical stoppage of the vessel with unirritating bodies; but if the embolus comes from a seat of inflammation, or is otherwise of an irritative kind, then other results follow which are due to infection. If, for instance, the embolus be from a seat of gangrene, the tissue of the part embolised generally soon passes into gangrene. It is thus that gangrenous dysentery sets up, by embolism of the portal vein, gangrene of the liver. We have a very typical example of this kind of occurrence in a specimen from a case of necrosis of the petrous bone, with gangrene of the dura mater and lateral sinus, and foul thrombus extending into the jugular vein; an embolus passed on from this vein into a branch of the pulmonary artery. The branch of the artery thus affected shows sloughing of its inner layers, which are dead, and separate easily from the outer layers, although the fœtid embolus had evidently only been a short time in the vessel, for it was unadherent.

But if the spot from which the embolus came was suppurating, then the effect is a suppuration in the part implicated. This suppuration, however, is complicated with the embolic passive hyperæmia we have above described, so that the suppuration is incomplete and consists rather in rapid breaking down of the tissues than in the formation of a large number of pus-cells, while the characteristic deep-purple, congested zone around the affected spot is much intensified. Some describe this as a true sphacelus of the affected part, but there is no necrosis and no foul decomposition of the patch affected in the suppurative form of embolic inflammation. Lower degrees of inflammatory quality in the embolic clot induce similar but slighter inflammatory conditions additional to the states we described as due to the mechanical obstruction. There are all gradations among such degrees. Under all these circumstances we now say the blood is septic, and micro-organisms are found in it. See further particulars under "Pyæmia."

It is a curious fact, and not easily explained, that the "embolic patches," as these portions of tissue suffering from emboli are called, are nearly all found at the surface of the organ affected. This is very constantly the case in the lung, spleen, and kidney, their chief seats. It may be that, central portions of organs being surrounded by vessels, a stagnation of blood is less likely to occur in them than in portions near the circumference where the vascular supply is on one side only.

We must now call attention to the fact that, so far as we have given explanation of all these changes, they do not arise from the act of embolism but from the presence of the clot, so that if we assumed the clot in the artery to arise spontaneously there, all the characteristic features of the "embolic patch" would follow in the field of the affected artery. Now, some very distinguished observers, without denying the occurrence of embolism, hold that many of these "embolic patches" do arise by spontaneous coagulation in the artery. They also believe that the blood may even coagulate suddenly in the pulmonary trunk, while the right heart is acting in apparent health. We have already seen that in old atheromatous arteries coagulation does occur, but this was a slow process and the clot was found in slender superimposed laminæ; also, we found that in very rare cases of general subacute arteritis clots will form in the arteries under circumstances that scarcely allow of our thinking them to be brought into the part, so that a certain ascertained basis of fact suitable for sustaining a belief in spontaneous coagulation within the arteries does exist, and some observers, as Dr. Murchison and Dr. Bristowe, hold this view strongly. They urge, concerning individual cases of supposed embolism, that there was no evident source of the embolus, and that there is no sufficient reason why coagulation of blood should not occur in the arteries as well as in the veins, so that they say the theory of embolism in such cases only shifts the difficulty from one part of the vascular system to another. It is equally certain that embolism occurs, and that spontaneous clotting of blood is met with in the arteries of old people. In every case that comes before us it will be well to give special attention to the evidence it individually offers in one direction or the other.

*Fat embolism.*—It is believed that in some cases death may be due to the plugging of the pulmonary arterioles with fat. This has been suggested to be the cause of the dyspnœa in fatal diabetes, and also especially to occur in cases of rapid death after the fracture of a bone, or laceration of a fatty liver. It is thought that oil globules may be carried from the injured medulla into the blood and then to the lungs, the vessels of which it blocks. We have little experience of it, but have been shown lungs where the contents of the vessels were rendered black by the addition of osmic acid, proving the presence of fat. It must be remembered that Dr. Handfield Jones has shown free fat in the smaller vessels arising from the disintegration of atheroma.

**Organization of Thrombus in Veins and Arteries.**—Little is accurately known of the organization of the chronic or inflammatory thrombus which we have just treated of in its natural connection with phlebitis and embolism, but some sections and drawings of a jugular vein running though a mass of large cervical glands show it to be full of organized thrombus. This must have been of slow formation, micro-sections through the vein exhibiting its walls pierced by vessels and a fibrous substance within it, permeated by rather wide vessels, coursing in all directions. We have also met with a case of similar closure of the longitudinal and lateral sinuses of the dura mater by vascular material ; the disease was evidently the result of chronic inflammation continued into the sinuses from the skull, for it showed serpiginous caries, which was probably syphilitic.

The behaviour of thrombus from sudden simple stasis, such as is found in the end of a ligatured artery as far as the nearest branch, has been well followed out experimentally, and some facts of great interest discovered. It is said that the white corpuscles of the blood change to stellate nuclei, such as characterise fibrous tissue, and it appears that vessels form in the clot generally before the tenth day. The vessels so formed are described as in open communication with the channel of the ligatured vessel, while they also branch out through the wall to become continuous with the vessels around. In this way the clot in the vessel grows vascular, then the vessels in it dilate greatly and the intermediate tissue wastes and changes, so that in the later stages we see the whole dwindled, but with a greater proportion of vessels than at first, resembling indeed cavernous tissue, so that the process is hence called cavernous metamorphosis. The knowledge of this process is chiefly based on experiments ; its application in pathology beyond the effects of surgical ligature is not yet known.

**Phleboliths.**—These are small earthy concretions found in the veins or their coats, and generally obstructing them. They appear to result from a deposition of clot or fibrin in the vessel, and a subsequent calcareous degeneration. They are met with mostly in the veins of the pelvic viscera, as of the bladder, uterus, lungs, &c., and in the rectum and spleen, also in the facial veins. The mass does not consist of true bony structure. Wedl, after carefully examining them, found elastic tissue in them, and also noticed a connection of some with the vein wall; hence, he thought it doubtful whether they are due to calcification of simple fibrinous concretions, but the fibrinous concretions in the peritoneum and pleura often show an appearance in their structure just like elastic tissue, and we do not think his evidence outweighs the constant relation of these bodies to the parts where stagnation renders it probable that the old and ordinary view is the correct one.

Besides these loose deposits the veins throughout a large part of their course may be affected by ossific changes, but these are not of the same character as in the arteries. In the latter, the sub-endothelial coat undergoes this calcareous alteration; but in the veins, is more on the exterior that the deposit is found, so that in the case of the arteries, supposing the iliacs and femoral were affected, they would retain more or less of their rounded shape, the coats themselves having undergone the mineral change; but if the corresponding veins were affected, the bony matter would seem to have been formed in the cellular coat, and in a very irregular manner, so that the original shape of the vessel would be lost, and if a section of the mass be made, it would be seen to be triangular, with the vessel, unless obliterated, running along one side. We have seen in the vessels above named large masses of earthy concretion on their surface, and if these be carefully examined, we find some trace of true bone texture in them. We before said that in the so-called bony changes in blood-vessels the calcareous matter might be called almost amorphous; but in some of this earthy matter on the exterior of the veins we sometimes see, on microscopic examination, a number of black spots at uniform distances, with lines from them, indicating a disposition to form lacunæ.

**Varicose Veins.**—A varicose condition of veins is very common in the lower extremities, known by their enlargement and serpentine course. The vessels, when examined, are found to be very much thickened in their coats, which may be as thick as those of arteries; sometimes they are dilated into large spaces resembling aneurysms of the arteries, but although they burst sometimes like aneurysms yet the pathological course of the two affections is very different. Veins tend to elongate and become tortuous, arteries to widen and dilate into great sacs; we have a large aneurysm of the portal vein, which formed a great blood tumour behind the liver. The portal vein, however, is, in many respects, like an artery rather than a vein. When the veins are about to dilate and become varicose the widening is found first just above the valves, then the valves give way and the vessel stretches, lengthening more than it widens, so that it is thrown into curves, which increase in number while they dilate in some parts more than in others. The cause of this affection is not positively known. It appears tolerably certain that pressure on the trunk of a vein will produce it, and therefore various surmises have been made as to the cause of the pressure; thus, pregnancy is constantly spoken of; but then, it must be remembered that varicose veins are more common in men; also, constipation is mentioned, but of this there is no proof.

Mr. Howse thinks too much stress has been laid on pressure as a cause, since there is seldom any history of it. He believes the condition to be more likely due to a local affection of the vein resulting from atheroma, and arising under strain. If inflammation followed such a strain, the coats would soften and give way. They would thus become varicose, and in time thickened.

**Hæmorrhoids.**—Formerly these were considered to be varicose veins of the rectum, but this is rarely the case; enlarged veins may be found in external piles; but we shall be surprised to see how much fibro-cellular substance is present in an ordinary pile, though it is highly vascular, and corresponding mostly to the fold of mucous membrane seen within the bowel. Very often piles have a true cavernous structure, being composed of cells which are filled with blood, and communicate freely with the hæmorrhoidal veins, their inner surface being continuous with the lining membrane of the vessels. They probably are formed in the first place from the veins, which distend and then break through into each other by destructive pressure on the intervening tissue.

**Varicocele** is the term applied to a varicose condition of the veins of the spermatic cord. The explanation of its more frequent occurrence on the left side is said to be the greater dependency of the testis, and the entry of the left spermatic vein into the renal vein. Not long ago we met with a case where the arteries were varicose, twisted together like a mass of worms, distended and bony. The varicosity may sometimes be due to changes in the veins themselves, as in the lower limbs (see 'Sexual Organs').

**Morbid Growths.** — *Tubercle* is almost unknown in the veins. *Cancer* does not occur primarily, but the veins may be involved in the disease. Thus, in those large masses of cancer affecting the lumbar glands, so often found in the abdomen or mediastinum, the coats of the vena cava may become implicated, and sometimes small sprouting fungous growths may be seen springing from the interior; or the vena cava may be utterly lost in the mass of a cancer.

# DISEASES OF THE MEDIASTINUM

*Acute inflammation* of the mediastinum sometimes occurs either independently, when an abscess is found beneath the sternum, or in connection with pericarditis, and passing up into the neck.

*Chronic inflammation* is met with leading to the formation of a tough fibrous tissue, which, extending upwards, may involve the vena cava and innominate vein, producing obstruction of these vessels. In many cases a pericarditis has been the starting-point, and associated with old thickened adherent pericarditis there has been a hard fibrous tissue proceeding up the aorta to the neck, and involving all the blood-vessels in its meshes.

**Tumours and New Growths.**—It is often very difficult to trace the origin of intrathoracic tumours, but many evidently have their beginning in the mediastinum ; the glands here are often affected, as in Hodgkin's disease. We meet also with lymphomatous, sarcomatous, and cancerous, growths. In one case we found a mass of syphilitic deposit binding all the parts together, and involving the vena cava with its branches.

# DISEASES OF THE LYMPHATIC VESSELS

We shall not occupy much time with this subject, which is almost entirely clinical; for *inflammation* of the lymphatic or absorbent vessels is best seen in the living subject, as after death the redness subsides, and anatomy throws little light on the early stages of the affection. When filled with pus, the vessels may be recognised by their knotted or varicose appearance ; they may so be sometimes seen in the broad ligament passing up to their respective glands in cases of puerperal fever

The most important affection of the lymphatics and lacteals is obstruction. This is not at all uncommon in tubercular disease of the mesenteric glands; of which we have many specimens. Vessels, of very irregular shape and distended, may be seen coursing over the intestine and passing along the mesentery to the glands. In some cases, where their contents are hard, it is probable that some tuberculous matter has penetrated them. The lacteals may constantly be found filled with chyle, without any obstruction being present, if the person die or be killed during the process of digestion. Sometimes, owing to obstruction of a duct, the chyle will collect and form a cyst, and thus small milk-white cysts may sometimes be found on the mesentery. In one very remarkable instance, which we saw here some years ago, such a cyst had burst, and the abdomen contained several pints of chyle resembling milk. Several similar cases of chylous fluid in the abdomen have been recorded, and will be mentioned under "Peritoneum."

Cases occur in which great varicose distension of the lymphatics of the thigh or trunk follow on some abscess, or other more or less obvious cause of obstruction. In such a case the lymphatics may form prominent cyst-like knots, puncture or rupture of which may yield pints of lymph.

To these new formations of lymphatic vessels, or a cavernous structure containing lymph, the name *lymphangioma* is given. Mr. Sydney Jones showed a case of this kind at the Pathological Society, where the legs were covered with soft swellings like varicose veins. When pinched they gave out lymph, and some being removed and examined were found to be made up of chambers composed of greatly distended lymph vessels. There was much hypertrophy of connective tissue around them. Dr. C. Fox also had a case of a boy who had a number of wart-like growths on his legs. These were soft and cystic, and when removed found to be dilated and varicose lymphatic vessels. A case related in the Paris journals describes a patient with a number of tumours on his body composed of a network of varicose lymphatic vessels. Lymphatic tumours are sometimes congenital.

We have recently met with several cases which make it probable that an obstruction to the course of the lymph stream through old glandular disease, scrofulous or otherwise, may weaken the tissue of the part obstructed, just as bad drainage spoils the soil and its produce. The evidence of this was the occcurrence of spontaneous low or ulcerative disease in such instances, limited to parts whose lymphatics led to obstructed glands, while the lymphatics themselves were full of pus. These occurrences we have found in the intestines from cretaceous degeneration of the mesenteric glands, and in the lungs and pleura from similar old disease of the bronchial glands. Mr. Hutchinson thinks the lymphatic vessels are often involved in syphilitic disease in common with the glands.

The thoracic duct is liable to obstruction at either end: if at its lower part it receives no chyle, and if at its upper part it becomes distended; and as no great nutriment can enter the blood in either case marasmus ensues. In tuberculous mesenteric disease this inanition occurs; but one of the simplest and most severe cases was a patient from whom the specimen was preserved; owing to some induration of the glands and surrounding cellular tissue in the left side of the neck, the termination of the thoracic duct had become obstructed; the consequence was a dilatation of the duct, and the most wretched emaciation of the patient that it was possible to witness. In a case of exophthalmic goitre, where the end of the thyroid gland passed deeply into the thorax, pressure by it on the end of the thoracic duct seemed to be the cause of extreme emaciation.

**Cancer.**—Besides the conveyance of cancer by the lymph stream to the lymph glands, cancer also is sometimes conveyed along the lymphatics so as to appear in knots in their walls. Mr. Birkett has placed in the Museum some beautiful dissections of cancer of the breast showing this. Koester believes himself to have proved that epithelial cancer spreads itself in the seat of its origin along the capillary lymphatics and takes its peculiar tubular forms from these.

*Chyluria.*—This is the case where lymph is mixed with the urine, rendering it milky; it has lately been found to be due to a connection between the lymphatics and uriniferous ducts perhaps from the passage of a parasite. The latter is the *Filaria sanguinis hominis,* not uncommon in the residents of India. It is supposed that the habitat of the worm is the lymphatic system, although it makes its way into the blood. In the case of chyluria it may travel into the kidney. In the so-called elephantiasis scroti, where there is a lymphatic discharge, these worms are constantly found. Dr. Stephen Mackenzie had a case of chyluria in the London Hospital, and he found the blood full of filariæ. It was remarkable that these were ordinarily found only in the night time, but when the patient changed the night to day as regards his habit of taking food, rest, and sleep, they were then discovered in the daytime. After the man's death, a large mass of glands was found extending along the spine, and the thoracic duct was much distended.

# DISEASES OF THE LYMPHATIC GLANDS

**Malformation.**—We would here mention an interesting, though theoretical, view of scrofula, which regards the characteristic glandular swellings of that disease as due to an original imperfection of the lymphatic glands, so that they are less permeable than natural, or liable to swell irritably, or otherwise to impede the flow through them on trifling accounts. It will be remembered how variable the glands are in their number, size, and mode of aggregation as we knew them in the dissecting room. A full consideration of these variations and their effects is still wanted.

**Inflammation.**—Inflammation of the glands is almost invariably due to irritating lymph from a disease in the part the lymph comes from; indeed, severe inflammation anywhere is apt to induce some sympathetic irritation of the corresponding lymphatic glands. Thus, in pneumonia we constantly find the bronchial and mediastinal glands very large, and pink or red in colour; but we should occupy too much time in detailing instances of this, which experience will soon furnish in plenty. If the inflammation be of a "benignant" or simple nature, the glandular irritation subsides with it and commonly is not noticed; on the other hand, if the inflammation be specifically poisoned, as in necrogenic pustules on the hands from dissection, or in soft chancre, then the swollen gland is apt to pass into a state of suppuration. It is curious and interesting to notice that some malignant inflammations have so great a tendency to attack the glands while others may leave these alone and spend their violence on the veins. We have twice seen inflammation from a malignant pustule or carbuncle of the face extend to the sinuses of the dura mater, and yet not implicate the glands at all. The implication of the glands in Oriental plague must be remembered; they play a very important part in its history under the title of buboes, and the facts observed prove that glands may be primarily affected in fevers.

Sometimes we have met a great abscess about the pelvic fascia, apparently originating in the glands from acute inflammation.

The minute anatomy of an inflamed gland is very simple, the small blood-vessels are distended with blood and the tissue is soft, showing vastly more of the lymph cells and less of the fibrillar network than is natural in the gland. But no question is more difficult to answer than that which asks how this simple change arises. Is it by wandering of lymph cells out of the vessels? Is it by multiplication of the lymph cells already formed? Is it by proliferation of the stellate cells of the meshwork? Each of these views has analogy in its favour. The most detailed descriptions, those of Billroth, favour the last view. We have seen these stellate cells containing many nuclei as if multiplying, but this condition was not so general as to persuade us that the great increase of lymph cells arose so. We rather think this action of the stellate cells of the stroma is connected with the increase of stroma and consequent induration which supervenes when the inflammation becomes chronic. It is most evident in *chronic inflammation*, which, in the glands, as elsewhere, differs anatomically from acute inflammation in producing increase of the fibrous stroma and comparative wasting of the cellular elements.

Particular glands are affected under special circumstances, generally from disease in the parts they receive lymph from, and thus the mesenteric glands are peculiarly affected in enteric fever. In this disease, associated with a peculiar deposit in Peyer's glands, the neighbouring mesenteric glands are also affected; these are very much enlarged, and, when cut open, are found red and soft. We have sometimes seen both the glands and the lymphatics full of effused blood in grave cases. The microscope shows the material to consist of a substance very rich in cells but having, on the whole, a close resemblance to the natural tissue of the lymphatic glands, so that it has hence been classed by Virchow with lymphoma.

It has been the subject of question whether this change in the glands in enterica is due to the disease in the intestine or is an independent affection of the glands. Our own experience leads us to think that it is certainly secondary; for, on the whole, it bears a close and direct proportion to the extent of the intestinal mischief, and it affects the glands that correspond to the diseased parts of the intestine. We have no experience of the enlargement of the bronchial glands, which some say belongs specially to typhoid fever. When they were enlarged at all in our cases there was always pneumonia present, and this, as we have already said, will of itself cause enlargement of those glands.

In *scarlet fever* the glands of the neck are sometimes very greatly swollen. The circumstances, in relation to the fauces and the anatomy of these glands, are identical with those of the mesenteric glands in typhoid. Sometimes these glandular swellings suppurate or slough, and produce great destruction of tissue; thus, we have seen the whole of the deep tissues of the neck exposed as if dissected from ear to ear, the hyoid bone lying quite bare on one side.

*Syphilitic disease of the glands.*—The indolent bubo of syphilis and its contrast with the suppurating bubo from non-infectious sores will be treated of in the surgical lectures. We must not be guided too absolutely by this distinction, for we have known constitutional disease sometimes to follow suppurating bubo. The minute anatomy of the syphilitic bubo has nothing in it characteristic; there is an increase of the lymphatic cells throughout the gland, as well as often an increase of the trabecular tissue. Such glands do not usually tend to become caseous like scrofulous glands. We sometimes find the glands implicated in late syphilitic gummatous masses in the mediastinum, neck, &c., but the disease in them has always appeared to be secondary in our examples of these cases.

**Morbid Growths.**—*Hypertrophy.*—We have already mentioned the extreme liability of the lymphatic glands to swell, and given instances of special diseases in which these swellings reach a large size, as in pneumonia, enterica, &c. Lymph-gland structure is so like some simple inflammatory products that the same description would roughly serve for both; we could say, namely, that either shows lymphoid cells lying in the meshes of a fibrillar material. This simple structure of the lymph glands must be especially noticed. If we consider it more precisely we shall find that the meshwork of a lymphatic gland has, perhaps, two special features; first, in having stellate cells at intervals connected with it, and second, in having a concentric disposition around the capillary vessels that supply the gland, the meshwork being separated from the vessel by an interval, which is the lymph path; the lymph-cells lie in small clusters in the meshes of the network. The stellate cells and the concentric arrangement are relied on in identifying lymph-gland structure, but these characters are certainly found well marked in tubercles, especially the large tubercles of the brain, and we have found them both in common inflammatory products.

If we duly notice how like lymph-gland tissue, which many regard as a permanently embryonic form of areolar tissue, is to inflammatory products, we shall be ready for a difficulty in interpretation of the enlargements of these glands, for new matter in the gland due to inflammation will have the same structure as the gland itself. There is positively no difference whatever between the normal lymph cells of the gland and the exudation cells produced by inflammation. Thus an inflammatory enlargement is structurally a hypertrophy of the gland, and we have to distinguish the two conditions by their circumstances.

When in the absence of inflammatory cause the glands are enlarged without any alteration of their natural structure, they are said to be *simply hypertrophied.* But simple hypertrophy of the glands is an unstable condition, and tends to pass into caseous degeneration so as to become "scrofulous," or else it gradually takes on the size and relations of tumour so as to become "lymphadenoma." It is clinically convenient to call large glands "hypertrophied" when they are persistent, and do not either suppurate or swell to great tumours. But there is no anatomical basis of distinction between the state of the glands in simple enlargement and that found in Hodgkin's disease, only that in the more moderate enlargement of simple hypertrophy the gland tissue is less altered than in Hodgkin's disease, and yet authors describe indurated as well as soft forms of hypertrophy. When this is allowed there is no distinction of hypertrophy from lymphadenoma, except in the circumstances and size of the growths.

*General lymphadenoma* (*lymphosarcoma, Virchow ; Adenie, Trousseau*), *Hodgkin's disease.*—There are some cases of glandular enlargement associated with anæmia, which are not infrequent, and which present so close a mutual resemblance clinically that it is convenient or even necessary to have a clinical name for them. We use the name Hodgkin's disease for this clinically recognisable state in justice to Dr. Hodgkin, who first drew attention to the peculiarities these cases present. Of late a great share of attention has been given to the microscopical detail of their structure and its kinship to other states, but this should not draw us off from that clinical identity of the cases which is recognised under the name Hodgkin's disease. It is very important to clearly see the relation between Hodgkin's disease and lymphadenoma. They are not different names of the same thing, but names of very different application; the former is the name of a peculiar disease of the glands, spleen, &c., while the latter is a name used for any tumour that has a lymph gland-like structure. Indeed, the name lymphadenoma is now used by the highest authorities say for cancerous disease of the skeleton, lungs, and heart, or for a malignant tumour of the kidney; so that it has become a histological rather than a pathological name, and has no *clinical* application like Hodgkin's disease. The latter is a general lymphadenoma of the glands so frequent as to require clinical recognition. This disease is generally not confined to the glands, it very commonly affects also the spleen, as well as the liver, and in both these organs consists of a development of a lymph gland-like tissue in the form of growths generally following the course of the lymphatics in the affected organs. The sections through these growths often have an angular outline and a whitish appearance that has been well compared to the appearances of pieces of suet scattered through the organ. Such are its more constant seats, but it may also be found in these cases in the walls of the intestine, especially the submucous coat, involving also the agminated and solitary glands, in the kidney, periosteum, dura mater, subcutaneous tissue, &c. In some cases it has one distribution, in others another, and the clinical symptoms vary somewhat in consequence, yet, in the cases where the lymphatic glands, spleen, and liver are chiefly affected, *i. e.* in Hodgkin's disease, the course of the disease is chronic, and its termination accompanied by marked anæmia and dropsy, no excess of white cells being present in the blood.

It was the wide cancer-like distribution of the disease, with the absence of those so-called "cancer cells," which were formerly regarded as characteristic of malignant growths, that drew attention to this peculiar affection. The microscopic structure was first clearly recognised by Virchow as essentially like lymphatic gland structure.

Subsequent writers, as Trousseau, or Cornil and Ranvier, have not added much to his description, but he unfortunately ignored the clinical importance of the disease, which they have recognised. We must notice that different examples of this disease, identified by the peculiar distribution and general appearance, show very different degrees of softness and hardness, and with these also very different clinical tendencies, the soft growing more rapidly, even becoming infectious to parts around, the harder forms growing more slowly and keeping more within their original bounds. But, soft or hard, the essential structure is very simple, and in short is that of lymphatic gland, the soft form having more cells and the hard more meshwork; but the cells resemble lymph cells, and the meshwork is that proper to lymphatic glands, the stellate cells of the meshwork and its concentric arrangement being also quite recognisable. The very soft forms too have often large polynucleated cells, while the meshwork is delicate, so that they become very like soft carcinomas, only that the cells are not epithelioid but lymphoid.

The resemblance to lymph gland is expressed in the names lymphadenoma and lymphosarcoma, the latter of which is from Virchow, while the former is adopted from Cornil and Ranvier. If the new formation were limited to the enlarged glands it would never have received so much attention, but must have been regarded as a simple hypertrophy of them, with varying induration or softening; but it is most important to observe that the likeness to the structure of glands is not limited to the growth in the glands, but is found in the growth in the spleen and in the liver, &c., so that the disease becomes more than a glandular hypertrophy, and it is still more curious to see that the softer examples may show a great power of contagion in their neighbourhood. Thus, in one of our cases the disease extended from the enlarged mediastinal glands across the pleura to the neighbouring lower lobe of the lung in which it spread freely. We here see a disease which begins as a simple swelling of the whole gland go on to transform other tissues around to the nature of gland in the most malignant way.

We frequently also see the trachea and bronchi invaded by the disease. It seems thus to hold an intermediate position between tubercle and cancer. The distribution resembles the former whilst its invasion and destruction of neighbouring parts makes it approximate to the latter. We occasionally meet with cases which are difficult to place in any category,—cases where the glands are much enlarged and have undergone caseous degeneration, and associated with deposits in various organs, which might be called either tubercular or lymphoid. We shall speak further of this disease under "Spleen." We have met with two cases of acute Hodgkin's disease in young lads. The disease lasted a few weeks, and was accompanied by febrile symptoms. The cervical and other lymphatic glands were enlarged to about the size of peas, and the liver, which was much enlarged, contained, like the spleen and kidneys, lymphatic deposits resembling tubercle to the naked eye.

Some have thought Hodgkin's disease may be traced to irritation of the glands through local disease in the parts they draw from, say caries of the ear setting up enlargement of a cervical gland and the enlargement extending from gland to gland. A successive enlargement of the gland is often traceable, but we do not know any facts showing that it is commenced from a simple irritation.

*Local lymphadenoma.*—Sometimes the same disease of the glands as becomes general in Hodgkin's disease, is found limited to one gland or a few glands or a packet of glands in one region of the body. This is most frequently seen in the neck; in such a case we have known the jugular vein entirely closed by the pressure of the glands. In many such cases the glands on section do not differ, except in size, from scrofulous glands. Virchow would call such glands scrofulous, however large they may be, whenever caseous degeneration occurs in them. Yet we owe to himself the evident demonstration that caseous changes occur in cancerous, syphilitic and, in short, almost every kind of tumour, so that the occurrence of the cheesy change seems an insufficient criterion. But we have seen that, when enormously large, these glands may evidence a scrofulous nature by setting up tuberlous pleurisy or peritonitis, which is a better proof of scrofulous nature. This occurred in an enormous cervical tumour already alluded to, which proved fatal by tuberculous pleurisy, and further it has happened not infrequently that, where these have been removed, the patient has subsequently died of phthisis or tuberculosis.

In other cases the tumours come so near other malignant diseases in their characters that they have formed part of the best examples the old "encephaloid cancer." As far as we have seen, primary cancer of the lymphatic glands is always lymphadenoma, which in this malignant form may be called *lymphoid cancer.* It is very frequently seen in the cervical glands, and also, not uncommonly, in the mediastinal and bronchial, forming part of the grave clinical cases known as mediastinal cancers (part, but not all, for some of these cases are truly carcinomatous, and then the glands are affected less than the surrounding tissue). Such primary lymphoid cancer is not infrequent in the lumbar glands, forming most of the cases known as "Lobstein's retro-peritoneal cancer." Another very important seat of the occurrence of lymphoid cancer is in the glands of the mesentery, which may grow to an enormous size; we have thrice seen such cancer extend down from the glands along the lacteals and reach the attached side of the bowel, spreading then around the bowel in a circle, yet widening, not narrowing it. This is the form which sometimes causes expansion of the affected part of the bowel.

*Scrofulous disease of the glands.*—This is distinguished from hypertrophy and lymphadenoma by its tendency to undergo caseous degeneration and by its alliance with tubercle. The gland enlarges by irritative hypertrophy, so that we find increase of the lymphoid cellular structure causing at first a slightly translucent swelling; this swelling, however, has a dryness such as characterises scrofulous swelling. This dryness signifies the dense aggregation of the new cellular growth in the gland and its want of freedom, so that it compactly compresses its own vessels, thus leading to gradual loss of vitality and caseation. The caseous change shows itself by all the elements of the texture gradually losing their sharpness and outline, wasting into fatty remains, and then these fatty relics may be entirely absorbed, which is a rare and the most favorable end. More often the caseous tissue breaks down into a curdy matter which may then dry into a pasty remainder which grows calcareous, especially in the mesenteric or bronchial glands, where the stony material in long intervals afterwards may undergo enlargement by accretion until a mass of stone as great as a walnut, or even much larger, may be found.

But instead of thus drying up there may arise a certain degree of more acute inflammation around, uniting the diseased gland closely with surrounding parts so that they become fixed as no simple hypertrophied or lymphadenomatous glands do. This inflammation may proceed to true suppuration and the abscess open either spontaneously or by incision, leaving a deep indolent "scrofulous" ulcer. We have, however, known these cold abscesses to recede without bursting, even when they have undermined the skin, and raised it as a thin cover over the contained pus.

It was the injection of the dried, caseous, or purulent remains left by such indolent glandular inflammation into the veins of dogs and other animals which produced, as it was thought, general tubercle, but the experiments have now been found to have been fallacious.

*Leuchæmic lymphoma.*—It is said by Virchow and others that in some cases leuchæmia depends on enlargement of the glands, though usually on enlargement of the spleen, and that the lymphatic variety is distinguished by a smaller size of the white cells in the blood. We have not yet observed any case of leuchæmia in which the spleen was not enlarged, yet the glands are sometimes enlarged with it. This fact suggests the theory that the glands may be secondarily affected from the leuchæmic condition. The structure of the glands in this disease is natural, and the only distinction from simple hypertrophy is the association with leuchæmia. We must mention that Virchow, in his description of leuchæmic tumours in the organs, does not unite them with Hodgkin's disease, though some have quoted him to that effect. The latter disease with him is lymphosarcoma, while leuchæmic tumour is lymphoma.

*Tubercle.*—Tubercle can be distinguished from scrofula in the glands only when it appears in its miliary form. Thus in cases of general tuberculosis we find the lymph glands often freely sprinkled with the same kind of tubercles as are found elsewhere in the body. Also in the bronchial glands corresponding to phthisical lungs, and in the mesenteric glands corresponding to phthisical intestines, we often find numerous miliary tubercles of quite characteristic appearance. Tubercle itself, so far as it has any structure, has a coarse resemblance to the simple structure of the lymphatic glands. In large tubercles of the brain we have seen the resemblance brought pretty close by the concentric character of the meshwork we have before spoken of as characterising lymph-gland structure. Regarding tubercle as a lymphatic growth it becomes in the glands a new formation of a little mass of gland tissue, and hence would be homologous in Virchow's sense of that word. Now Virchow regards tubercle as a heterologous and malignant lymphoma. On his view malignancy goes with heterology, that is, with a non-resemblance to the tissue the new formation occurs in. To explain this difficulty, Virchow regards tubercle as not commencing from the proper tissue of the gland, but arising in the septa, &c., of the gland. We have, however, not been able to see anything to substantiate this complex view: rather we have found in tubercle of the glands a low structure of fibrils and lymphoid cells spreading through the glandular structure, and the resemblance of this low structure to gland was due to the fact that both had lymph cells and neither had very much else in which to differ from the other, excluding the vessels, &c., of the gland, which are not taken into account in Virchow's statement; in short, the intricate difficulty thus created is an altogether unnecessary refinement of a simple subject.

*Relation of tubercle to scrofula.*—Whether scrofulous disease, with its general or widely spread large patch of caseous change, is the same as tubercle with its smaller miliary grain is a question that has continually been asked, but at the present time is distinctly settled by the presence or not of the characteristic bacilli. True tubercles may be found primarily in the glands or as secondary deposit through infection. Eve inoculated from strumous glands, and produced internal tuberculosis; a similar fact is seen in the occurrence of tuberculous meningitis being the secondary effect of caseating bronchial and other glands. The scrofulous glands of which we have been speaking may have been tuberculous or merely the result of an inflammatory process which has terminated in caseation. It may be remembered also that, so far from miliary tubercle degenerating into a caseous material, there is an opinion held by some that the opposite is the truth, and that, as in the experiment just named, tubercle is the secondary form of which the so-called scrofula is the primary form.

*Lardaceous disease of the glands.*—This is frequent when lardaceous disease is present in the abdominal viscera; the cervical glands are especially liable. The disease does not cause great swelling; the organs harden and become pellucid; iodine brings out the characteristic walnut-wood coloured stain. Microscopic examination shows that the small arteries are, as usual, first affected; they become swollen and lustrous. The change then extends to the lymph cells, which swell, and often unite into masses of glistening lardaceous matter. (For a more particular description of the change see the description of it under "Spleen.") Cornil describes a hyaline degeneration where there is no iodine reaction.

**Cancer.**—One of the first things by which a cancer in any part proves its malignancy is the infection of the corresponding lymphatic glands, and thus secondary cancer of the lymphatic glands is extremely frequent; we were going to say almost as frequent as cancer in all other parts together, but not quite so frequent as this would imply, for some cancers in vital parts kill before there is time to infect the glands; and other cancers, especially the malignant sarcomata, are remarkable for their disposition to extend to remote organs, without implicating the lymphatic glands. Perhaps it was because of this frequency that it was thought by some that cancer of the lymphatic glands was always secondary; but we feel sure that primary cancer of the lymphatic glands does frequently occur, and then, as we just now said, has the characters of lymphoid cancer, or soft lymphadenoma. We need not enumerate the kinds of cancer that appear secondarily in the glands, suffice it to say that they all do so.

**Arrest of Pigments and Poisons in the Glands.**—The spaces between the tissues are continuous with lymph vessels, and these lead up to the glands; and by the way thus open foreign substances in sufficiently finely pulverised, or rather molecular, state reach the glands. Thus some of the cinnabar and carbon used in tattooing a man's arms will be found in the axillary glands, lodged close to the afferent vessels, for the gland tissue acts as a filter, and stops the foreign substances. But this process is exhibited on the largest scale in all of us, whether we will or not, although we might object to be tattooed. The seat of it is the lungs and bronchial glands. Pigmentary matter, such as the fine dust in the air, enters the lungs; the cilia of the trachea and bronchia waft back most of it, but some gets down to the air-cells, and penetrates the tissue. We know how the living animal membranes have recently been proved to be permeable to solid particles; thus we can understand how the dust gets into the lymph paths. It can be traced along the course of the lymphatics, beside the arteries, in the tissue of the lung, and at last it is found accumulated at the entrance of the lymphatics into the bronchial glands. In persons who have breathed pure air for a long while, or in young persons, the amount of this pigment may be small; but in old inhabitants of large cities, and *a fortiori*, in those engaged in very dusty occupations, such persons as coal miners, needle grinders, millstone grinders, workers in hair, tobacco, &c., we find a vast accumulation of pigment in the glands—primarily of course in the lung. Some think the pigment is principally hæmatoidin pigment, due to irritation and extravasation of blood, which subsequently undergoes changes; but the mesenteric glands are very liable to irritation, and yet do not (except very rarely) become pigmented, so that the black matter appears, indeed, to be the dust breathed.

We have already alluded to the probable effect on the nutrition of the lung which the obstruction of its lymphatic glands must have.

It is not only inorganic matters that are thus stopped by the glands. Animal poisons likewise are arrested by them, and this, indeed, appears to be the chief part of their function in pathology. Thus syphilitic poison rarely goes beyond the first tier of glands. We have a drawing made from the axillary gland of a lad with leprosy, whose skin was in a frightful state of disease, and we see from it how at the entry of each afferent duct there is a patch of yellow leprous matter having exactly the same structure as the leprous disease which was in the skin. It had existed in the skin for seven years, and yet it had made not one tenth of an inch progress in the gland ; thus the gland tenaciously prevented admixture of the poison with the blood.

# DISEASES OF THE NERVOUS SYSTEM

## BRAIN

Although it is convenient, in studying morbid anatomy, to take the various structures separately, it should be remembered that in the larger sense of pathology they should be considered together, since it is often impossible for one structure to be affected without another. Thus, the brain and its membranes are frequently diseased at the same time, and with them often the cranium; but it is more convenient to study these part by part. The same remarks hold good in other organs, as the lungs, where, for instance, in former times, an inflammation was meant to include all the tissues in the chest; but now, by greater refinement, we speak of pleurisy, pneumonia, and bronchitis as distinct; but in so separating diseases for the sake of study we should remember that practically they often all exist together, and that such combinations are often more frequent than the individual affections; thus probably an acute inflammation of the chest, involving all the tissues, is as frequent as one affecting these separately, and the same also in chronic disease, as phthisis. In speaking, therefore, of the membranes and brain separately we must remember that practically their diseases are not thus always rigidly and anatomically distinct, and as a matter of fact all the symptoms of meningitis are referable to the substance of the brain and not to the membranes.

The eye should be familiarised with the general outline of the brain so as to see at once what portions of the organ are affected by disease, more particularly the frontal, parietal, and occipital lobes, with the well-marked fissures of Sylvius and Rolando. The observer should at once be able to recognise the parietal or motor region with its ascending parietal gyrus behind the fissure of Rolando, and in front of it the ascending frontal gyrus with the superior, middle, and inferior, convolutions which are given off from it. Then he should observe the general appearance of the brain as regards its plumpness or wasting; changes in colour and consistence, both on the surface and when sections are made; also the state of the vessels, their rigidity, and whether filled with clot; also particularly the appearance of the membranes as regards thickness and opacity; also whether fluid be beneath them; also the amount of fluid in the ventricles. When examining by the microscope he should observe all departure from health in the large cells, whether they are smaller, darker, or have lost their processes; also the nerves, whether they are partially or entirely destroyed, and the presence of new connective tissue, and other conditions which will be mentioned in detail.

**Malformations.**—These so usually affect the brain and all its coverings together that it will be convenient to treat of them while speaking generally of the brain as a whole.

Sometimes the lateral halves of the brain coalesce, or rather are never separated, so that the hemispheres are conjoined and the eyes develop in a united state, hence called *cyclops* or *monoculus.*

The brain or spinal cord may be absent or very imperfectly developed, *anencephalus, microcephalus,* &c., or the ventricles of the brain and cord, or of either alone, may be much dilated (*congenital hydrocephalus, congenital hydromyelus*); but the most important malformations are those wherein the head attains to a fair degree of development, but parts naturally within the skull protrude as tumours outwardly. We have already mentioned how often the laminæ of the spine are deficient and the meninges protrude, forming a cyst in the back; in the same manner, from an unnatural opening in the cranium, the cerebral membranes may escape and form a sac, in which may be a part of the brain itself.

The usual position for such a deficiency of bone is either behind, in the occipital region between the fontanelle and the foramen magnum, or low down in the forehead. Of seventy-five examples collected by Mr. Zachariah Lawrence fifty-seven were occipital and seventeen frontal, but such protrusions sometimes occur at the sides of the head, and in other cases the deficiency is about the sella turcica, usually just in front of it, the tumour projecting down through the roof of the mouth.

In one specimen, where the calvaria has been removed, we can see the hole within, and the membranes passing out to form the external sac. In another preparation, we can better see how the cyst is formed: the arachnoid appears free, but the dura mater is lost on the external covering of integument. If the protrusion arise simply from a bag of membranes containing fluid, the affection is called *hydromeningocele;* such a case we had an opportunity of lately seeing in Mary Ward. The cyst protruded from the back of the head, and was quite transparent; it spontaneously sloughed, the fluid escaped, and when the child left, the wound was healing. When the tumour contains brain structure as well as fluid the affection is simply called *hydrencephalocele,* or if there is no fluid it is termed an *encephalocele;* if the brain enters, there is usually a prolongation of the ventricle within the extended portion of brain.

A very good example of hydrencephalocele is seen in one of our drawings representing an infant, from the posterior part of whose head a tumour hangs down almost, if not quite, as large as the head itself. In the middle of the occipital bone is a round opening, and it appears as if half the brain had been squeezed through this, so that only the anterior and part of the middle lobes of the brain were within the true cranium, which was very small from the frontal bone being pressed down; the remaining portion was in the sac behind. The ventricles, in like manner, were divided between the two, the sac containing their hinder part with the choroid plexuses; the posterior lobes were adherent to the membranes which formed the sac, and corresponded to the parts of the tumour on which pressure had been made in supporting the head, and which were sloughing, the corresponding portions of brain being also softened; the cerebellum was within the cranium, as usual. This shows that such a condition of head is incompatible with life, if it only be from the want of a protecting case. But in an instance given by Mr. Hutchinson the cerebellum was included in the tumour; and in one given by Dr. Murchison there was no cerebellum at all. Minor degrees, however, of encephalocele will allow of life, so that their subjects may grow to adult age. These tumours are usually met with at the root of the nose, between and above the eyes, or more rarely in the occipital bone. When a congenital tumour is found high up in the neck, it is sometimes difficult to say whether the brain or upper spinal region is affected. In such a case, where a cyst had existed for many years, Mr. Solly describes its neck or peduncle as becoming impervious—this is, it underwent a spontaneous cure—and then the sac was removed.

Some malformations are probably the result of inflammatory conditions during fœtal life. Thus we may see the falx cerebri deficient at its anterior part, and the two hemispheres morbidly adherent. This, probably, is due to some inflammatory action at the time mentioned; for we find it in connection with atrophy of the brain, thus showing it not to be a simple arrest of development of the falx, but a consequence of a more general morbid action. As to the probability of the fœtal brain inflaming, we have found inflammatory adhesions and effusions as early as the fourth month of fœtal life, and we shall shortly speak of the frequency of cerebritis in the syphilitic fœtus at full term. As a malformation, we sometimes meet with a dilatation of the sinuses.

## DURA-ARACHNOID

**Hypertrophy.**—In cases where the bones are thickened, as in the specimens we exhibited in the first lecture, the dura mater is often much affected in the same way. This thickening has especially been found in cases of epilepsy, and in some cases of chronic mania; also, wherever the bone has been the subject of ostitis.

**Atrophy.**—The dura mater may often be found thinned in parts, especially along the middle line and near the Pacchionian bodies.

**Connection of Dura-arachnitis with Injury and Disease of the Skull.**—This membrane is essentially double; its outer layer corresponds to the periosteum of bone, and belongs intimately to the bone, so that it is affected with it, and not with the organ inside ; indeed, it affords a valid protection to the brain, and its outer surface may be diseased to a very great extent without this organ at all suffering. The dura mater itself is not primarily affected by great inflammatory processes, but only in connection with the skull, and thus it is not found diseased except in cases of fracture of the cranium or necrosis of the bones, &c. In the former case, it may be injured at the same time with the bone, be lacerated, and afterwards slough through the direct injury; or, at a subsequent period, after portions of cranium are removed, and the surface of the membrane exposed, it may become involved in the softening inflammatory processes. It is more rare to get suppuration about the dura mater from injury without fracture. In cases of disease of the ear and temporal bone the contiguous dura mater may become involved, changed to a dark colour, and slough ; or suppuration may rarely occur between it and the bone, lifting it off the bone in the form of an abscess. In any of these cases there is danger of implication of the sinuses, and consequent purulent infection. We have seen, however, meningitis set up by ear disease without evident affection of the dura mater. The more chronic inflammatory processes are seen in those cases, especially in syphilis, where large portions of the calvaria have come away, and the dura mater is exposed : in these we may find the outer surface of the membrane covered with large patches of lymph, which form hard granular layers, producing much thickening of the whole membrane. It is remarkable for how long a time the inner serous surface remains unaffected in such a case.

In some instances, after injury to the bone, the dura mater becomes adherent to the parts beneath, and if the chronic inflammatory process continue, the brain may be involved. Thus, in a man who, since receiving an injury, had suffered with epilepsy, the bone was much thickened by ostitis, as well as the dura mater beneath it, which was inseparably connected with the underlying convolutions.

**Acute Inflammation of Inner Surface of Dura Mater—(Dura-arachnitis).**—Recent anatomists have almost explained away the arachnoid, declaring the "parietal layer" to be an epithelium on the dura mater, and the "visceral layer" to be only the smooth outer face of the pia mater. But the space known as the arachnoid cavity, like the other great serous surfaces, offers opportunities for the formation and accumulation of morbid products ; and these products arise under such special circumstances, or else are so characteristic, that it is necessary in pathology to recognise the serous nature of the arachnoid surface.

Nevertheless, what the modern anatomists have said of the relations of the arachnoid to the dura mater has a certain interesting analogy with what we shall now see ; for while the anatomists say the parietal arachnoid is only the inner epithelial face of the dura mater, we, as pathologists, must declare that the inflammations of the arachnoid occur only through the medium of injuries to the dura mater; and, again, when anatomists deny to the visceral layer any special nature, and regard the pia mater as the true and proper membrane, of which the arachnoid is a mere superficial face, we, as pathologists, must say that this visceral arachnoid has equally no distinct pathological existence, and never spontaneously inflames, nor even in spontaneous inflammation of the whole covering of the brain does it show any activity; for while the deeper vascular layer of the membrane may fill the interval between itself and the arachnoid surface with lymph, there is not any noticeable formation on the free arachnoid surface.

We find students who have no practical information on the subject suppose that, as in pericarditis, pleuritis, &c., the inflammatory products are poured out between the serous surfaces; so, in the case of the membranes of the brain, or in arachnitis as it is called, the inflammatory products are found in the arachnoid space. This is, however, wrong; in idiopathic inflammation, the exudation is from the pia mater beneath the visceral arachnoid, although there is some slight secretion from the other side; and thus we prefer using the more general expression, meningitis, in the idiopathic inflammation of the membranes of the brain. In true arachnitis, where the exudation is between the serous surfaces, as in pleurisy, &c., we believe the disease invariably originates from the dura mater and skull. We have never seen extensive exudation in this interarachnoid space without an injury to the head, or caries of the internal ear, or syphilitic caries or other cause of suppuration in the bones, or else through meningitis from bedsore, or from disease of the spinal column extending up to reach the brain; and thus we speak pretty confidently on the point. We have never ourselves known such an affection as this simple arachnitis spring up as a spontaneous disease, and therefore, when we meet with it we always look for some mischief outside. When arising in this way, the interarachnoid effusion is often purulent, and so copious that it may pour out when the dura mater is removed. It may form a circumscribed abscess, limited by adhesions around; cases are recorded of successful opening and cure of such abscesses. This purulent effusion more rarely extends to the base. It is found principally on the side of the fracture or injury, being often prevented from extending to the other side by the falx. Thus, we sometimes meet with a unilateral arachnitis, and if the inflammation reach across the falx, the amount of effusion is less on the far side. The inner surface of the dura mater is in these cases covered with lymph and pus; with this effusion there will generally be also an exudation of lymph beneath the visceral arachnoid, as in idiopathic inflammation, forming an opaque sulphur-coloured layer in the subarachnoid space, over the convolutions; but whenever there is lymph in the arachnoid cavity we must look for a cause externally in the dura mater and skull.

Although we say thus confidently that pus in the arachnoid cavity is constantly an indication of injury, or disease of the bone or dura mater, yet we find a few examples of pus within the arachnoid sac, as an extension from the spinal canal. This has been seen by Husch in a few exceptional cases of epidemic cerebro-spinal meningitis, and Dr. Murchison has given a similar case. Dura-arachnitis, as already mentioned, sometimes occurs from blows on the head without fracture.

*Thrombosis of cerebral sinuses.*—We have already referred to the occurrence of this condition when on the subject of Veins, and stated that we have met with it in children and occasionally in adults. In the latter the patients have generally been young women, and have suffered from hæmorrhage, or been anæmic, so that in all probability the coagulation has taken place from some change in the blood itself. In one case, after about a week's illness of intense headache terminating in convulsions, the patient died, and the longitudinal sinuses and lateral sinuses were filled with adherent fibrin, and all the veins of the pia mater were full of coagulated blood. The surface of the brain was turgid from congestion. Four other cases, of which we have notes, occurred in young girls who had suffered from anæmia. Death took place in a few days after the onset of the cerebral symptoms; the sinuses were filled with a firm clot, and there were extravasations into the brain substance. Bright gives a drawing of the brain of a child who died of this affection.

**Effusion of Blood or Meningeal Hæmorrhage.**—When blood is met with in the interarachnoid space, it may have proceeded either from the vessels of the dura mater without or pia mater within. It rarely originates from without, since rupture of the meningeal arteries through injury is attended generally by a flow of blood external to the dura mater, between that and the bone; while other causes, such as an aneurysm on a meningeal artery, or any other bleeding tumours, are rare pathological curiosities. On one or two occasions, however, where, after a blow on the head, blood has been found covering the arachnoid, and no injury to the brain or dura mater could be discovered, it was considered that the source must have been some of the vessels in the dura mater, because this membrane is more immediately concerned in shocks to the bone. We have seen a few such cases; in one a boy died after a blow on the head from a stone thrown at him. There was no fracture of the skull, and no sign of injury to the brain, but much blood was in the arachnoid cavity; also we remember a similar accident to a blind man, who fell on his head, and several such cases are recorded. These accidents generally affect elderly people through falls. It has often been stated that meningeal effusion occurs more frequently in the intemperate. The blood sometimes evidently originates in the pia mater, and may have come by spontaneous hæmorrhage, or from an injury. Thus in fractures of the skull, if the brain have been at all bruised, and the arachnoid torn, some blood will be found in the arachnoid space, as well as under the pia mater. This is of everyday occurrence; sometimes in cases of fracture, blood is effused into the arachnoid cavity without the dura or pia mater being evidently torn, and then some observers believe it has come from rupture of the veins that cross from the brain to the sinuses. Interarachnoid hæmorrhage arising spontaneously is not very common. We have seen it in a child, who had died of convulsions in hooping-cough; it would then be called *meningeal apoplexy;* it resembles the ecchymoses of the eye in the same disease. In all the other cases in which we have witnessed it (and they have not been many), the patient has had Bright's disease. In some of these it was quite unexpected, and had evidently happened only a short time before death; on removing the dura mater, a thin layer of recent black coagulum was found partly covering the hemisphere on one side; its source must have been the pia mater, though it was impossible to find the ruptured vessel.

Dr. Goodhart, who has collected a large number of cases, sees no reason to look necessarily for the source of hæmorrhage in aneurysm or accident seeing that the vessels themselves are diseased, and that the same causes lead to hæmorrhage on the surface of the brain as in its substance; for in most cases not only is the vessel diseased but the heart has been hypertrophied, and the kidneys degenerated. In a child with endocarditis the hæmorrhage was probably due to infarction. It has also been observed in newly-born children after difficult labour.

**Chronic Hæmorrhagic Dura-arachnitis.—Pachymeningitis.—Hæmatoma.—Changes connected with old hæmorrhage.**—We are now upon much debated ground, namely, that which concerns the presence of certain layers of false membrane with more or less altered blood in them, attached to the dura mater, sometimes so closely as to thicken it; sometimes with effusions of recent blood between the layers of false membrane, so as to make a blood tumour (hence called *hœmatoma of the dura mater*). Blood, recent or old, coexists in them with false membrane, such as we see in serous inflammations, and the question is whether the blood causes the false membrane by inflammation, or the false membrane, which is vascular, bleeds, producing the blood. We will first take one certain step, which cases that come under our own observation place beyond a doubt, proving that effused blood will produce these layers. We have seen several examples where the prominent lower parts of the middle and anterior lobes of the brain showed old bruising so exactly in the manner constantly seen in cases of severe injury to the head that there could be no doubt such an injury formerly had been suffered. This was, in two of the cases, rendered certain by the presence of an old, healed, depressed fracture of the left parietal bone. Now, in these cases, a considerable extent of the dura mater, reaching sometimes to the vertex, had a lining consisting of an adherent yet separate layer, identical with the so-called pachymeningitis; that is, a brick-dust coloured layer, which, under the microscope, showed a quite beautiful structure—the characteristic structure of pachymeningitis. It was full of wide capillary meshwork of long tortuous vessels, which in many parts showed varicose dilatations; supporting the meshes was a delicate texture of spindle-cell connective tissue, in which were numerous crystals and glomerules of hæmatoidin, which gave the red colour to the membrane. These membranes were long ago described accurately by Wedl, without their connection with hæmorrhage being suspected. The examples just given will show that an old traumatic effusion of blood will cause the false membrane in question; and we must therefore believe that such membranes are so caused, until it is equally clearly shown that they can arise by spontaneous inflammation. The other mode of origin for these membranes advanced by Hodgkin and subsequently by Virchow, and generally accepted abroad, is this: the inner surface of the dura mater is thought to produce by irritation a layer of inflammatory false membrane, which becomes highly vascular. As stated by Rindfleisch, this view goes on to say that new layers form on the surface, and are again highly vascular, while the old layers beneath contract on and obstruct the returning circulation through the vessels of the superficial layer, so that these burst, causing effusion between the layers, leading thus to larger or smaller effusions of blood. The larger effusions may distend the false membrane into the form of a cyst, but the smaller will be found long after as old altered blood-pigment; some go so far as to point out the share of the epithelium in the production of the original false membrane, which is said to be evidently considerable, as its cells are swollen much around the edge of the new formation. But long ago Mr. Prescott Hewett had traced the whole process in many cases to an effusion of blood, and shown how this, in course of time, assumes the form and structure of a serous cyst; and there is no doubt that if, from injury, blood be effused between the arachnoid surfaces, it forms a thin layer over one or the other hemisphere, and coagulates; soon the fluid part is absorbed, and the fibrinous layer undergoes organization, while the colouring matter passes through the usual changes; the colour alters to a brown hue, the corpuscles are broken up, and the hæmatin crystallises. Perhaps, however, both methods of formation occur in different cases. Yet we think that hæmorrhage is generally the first step in the process, though probably bleeding into the new membrane happens afterwards, as described by Virchow. In a case we examined not long ago, where death occurred three weeks after the injury, a thin brown layer covered the hemispheres and inside of the dura mater in this manner; and in a case eleven days after injury there were distinct ochry layers. Mr. Prescott Hewett says he has found a thin and smooth membrane, like a serous membrane, in twenty-five days after injury. The crystals of which we speak are named *hæmatoidin*, and are formed from the hæmatin;

they are of a deep ruby-red colour, and in shape oblique rhombic prisms, sometimes a little more irregular, and if seen for the first time they might perhaps be mistaken for uric acid. It might be important to know at what time such crystallization takes place, so as to ascertain the duration of the effusion; but of this we cannot speak more positively than that they may be made in a short time artificially; but in the human body we never saw them within three weeks after a sanguineous effusion; in recently poured-out blood the hæmatin is in irregular granular masses. It is after a longer period, as several years, that such a film of blood will form a distinct vascular membrane, such as we have described.

When the effusion of blood is large, and the inner and outer surfaces of the clot thus widely separated for a long time, a distinct layer forms on each face of it, which may remain permanently separate, so as to produce a cyst, resembling the old apoplectic cysts within the brain which we shall soon describe.

These cysts may thicken enormously. Thus, in one case we met with in an idiot boy some time ago, the cyst, which covered the whole upper surface of one hemisphere, was nearly as thick as the dura mater, to which it adhered more firmly than to the arachnoid. Its contents consisted of a whitish paste, glistening with cholesterine. Some such cysts have been met with presenting no sign or little signs of blood, but containing a watery fluid. They have been called by Virchow *hygroma*. There is a very good specimen in the Museum, and a good drawing of a cyst in the Sixth Volume of the 'Transactions of the Pathological Society.'

**Syphilitic Inflammation of the Dura Mater.**—This is often met with; the outer surface of the membrane and the bone are generally affected; at the same time the disease tends strongly to extend to the inner surface, and cause adhesion to the brain at the affected spot, or it may excite suppurative arachnitis. The disease produces a fleshy swelling, pink or red from congestion and vascularity of the new products, and early showing a caseous patch in the centre of the affected portion. Of its occasional resemblance to sarcoma we shall presently speak.

**Adhesions.**—In children and old people the dura mater is naturally adherent to the skull. In young infants we have often found great difficulty in separating the two, and in old people have been obliged to use considerable force to remove the calvaria along the median line and over the frontal bone; and even then the membrane has been most likely torn, and portions remain adherent to the interior of the skull. We expect to find these adhesions in the young and old, but in the adult they are unnatural, and show disease. Thus we may meet with them in all cases where the bones are thickened, as in the instances before mentioned, but especially in those cases where a distinct ostitis has resulted from a blow; in any such examples the membrane may be adherent, and in exceptional instances, where lymph has been formed on the inner side of the dura mater, it may be found also closely connected with the brain.

**Separation** of the dura mater from the cranium usually arises from effusion of blood, owing to laceration of a meningeal artery, generally the middle, in cases of fracture of the skull, perhaps more often in old people whose meningeal arteries run in deeper grooves of the bone; sometimes, also from laceration of its great sinuses. Rupture may also readily occur from a blow without fracture of the skull. Blood is poured out between the dura mater and the cranium, forming a tumour, which presses on the brain; the clot in such cases is curiously firm and hard, and of a dull purple colour. This is due to the compression of it between the bone and dura mater, which causes absorption of the more liquid parts. It is to these cases that trephining is especially applicable, and such clots will generally be found about the large branches of the middle meningeal artery. It is not often that they exceed an ounce, while the clot of fatal apoplexy is generally three to four ounces; but there is usually a good deal of blood effused inside in the arachnoid and subarachnoid spaces at the same time, hence the compression.

The dura arachnoid is sometimes lacerated in injuries, so that the brain protrudes forming a *hernia cerebri*. Often, however, it is after some days' inflammatory action in and beneath the dura mater that the perforation occurs spontaneously or by operation. We have sometimes seen a laceration of the longitudinal or lateral sinus causing fatal hæmorrhage in fracture of the skull. For example, at the top of the lambdoidal suture in a specimen of a skull a Wormian bone is so turned round, and its sharp point turned in, that it was thrust into the longitudinal sinus, yet the little bone is so fast set in its new position that you cannot pull it out. Sometimes a simple fissure along the skull tears open the sinus, and then fatal bleeding may occur, if there is a free external wound.

**Morbid Growths.**—Among the most frequent changes in the dura-arachnoid is a production of *bone.* This may proceed until the whole structure is changed into a bony case, and appears like one calvaria within another. In lesser degrees it is very commonly met with, and especially in the falx major, which is its favourite seat. In a case lately we found a piece like a tooth, and on examination by the microscope it was found, like other similar specimens, to be true bone, the lacunæ and canaliculi being admirably displayed. We have said that the so-called ossifications of the heart and blood-vessels consisted merely of very simple earthy concretions, so that they should rather be called cretifications or petrifactions; whereas now it may be remembered that the ossifications of the membranes of the brain and spinal cord consist of true bone.

In speaking of *cancer* of the skull we stated that in many instances it appeared to commence in the dura mater, and then penetrated the bone. Its existence may not be suspected until an attempt is made to strip the dura mater from the skull, when the cancerous deposit is opened; it is then seen to form round soft patches on the membrane, and on the corresponding surface of the skull the bone is found eaten away in its internal table, and occupied by the same cancerous material. Although most authorities agree in describing these multiple round tumours on the outside of the dura mater as sometimes belonging to bone and sometimes to membrane, yet we think these tumours more properly belong to the former, as we shall see by observing, first, how frequently they coexist with general cancer of the skeleton, and secondly, how close a resemblance there is between all the cases. Sometimes single tumours project inwardly towards the brain, but being found also in the substance of the membrane implicating the bone, and pressing inwards, a question may arise whether in such cases the bone was not first involved. The growths that spring from the inner surface of the dura mater, as they rise towards the interior of the skull, take within it the form of low protuberances; one of these may swell at the most prominent part so as to acquire a constricted appearance at the attachment, and we have found such a polypoid tumour growing across the arachnoid cavity into the substance of the brain to some depth. Such projecting tumours vary from a fibrous to a fleshy consistence. Their microscopic structure is commonly that of well-characterised *sarcoma* or *fibro-sarcoma*, the consistence being more firm the more fibrous the structure; some examples have a mucous intercellular substance, and hence would be called *myxoma*. These, however, have generally affected the dura arachnoid of the spinal cord. An interesting form of tumour is described by Virchow under the name *psammoma*. These tumours, he says, affect the parietal dura mater principally as hemispherical excrescences separable without difficulty; they are tolerably firm, of reddish or marrowy white colour, and on section show a quantity of sand-like grains, which under water fall out or remain connected with the loose vascular meshwork of which the substance is composed. He connects the sandy nature of these tumours with the sand grains found naturally on the dura arachnoid about the sella turcica and elsewhere. They are made up of spheres or connective-tissue rings, and in these is deposited calcareous matter; without the sand the tumours would be called by some endothelioma. We have in our Museum a specimen where the tumour is of large size, springing from the anterior junction of the falx with the tentorium cerebelli. Dr. Goodhart called it "psammo-sarcoma."

All these tumours show a predilection for the neighbourhood of the body of the sphenoid, and next for the falx at its anterior part. As a rule, internally projecting tumours come more from the middle region of base of the skull than any other part of it. This leads us to remark that such tumours are often difficult to distinguish from syphilitic growths, which also are mostly found about the sphenoid, because these too are liable to undergo a fatty or caseous degeneration such as we not infrequently meet with in sarcomas; sometimes it is only by a careful consideration of the attendant conditions that we can tell syphilitic gumma of the dura mater from sarcoma.

The dura mater is, as we know, smooth on its inner face, being lined by the arachnoid, which, however, here consists of little more than a layer of epithelium. In considering growths from the inner surface the question comes, do they arise from the dura mater or from the arachnoid? This question acquires great interest with respect to certain examples of epithelial cancer, which have been met with generally in the occipital region. But such a question is very difficult to answer; epithelial cancers usually belong to surfaces where a thick coating of epithelium is found, as the skin or mouth, &c. The arachnoid is, we believe, the only serous surface which has been known to give rise to epithelial cancer; no one has yet shown what share the epithelium has in it. We have seen a small villous growth from the dura mater over the basilar process. The inner surface of the dura mater, like the outer surface of the pia mater, produces Pacchionian bodies.

*Hydatids*, which are occasionally found in the brain, may also develop within the arachnoid cavity, as in one case which occurred some years ago at the Surrey Dispensary. On removing the dura mater two or three cysticerci fell off from the surface; they were loose, or only slightly connected with the arachnoid. We have an old Museum preparation, showing a cysticercus from the surface.

## PIA-ARACHNOID AND SUBARACHNOID SPACE

**Congestion.**—We must warn the inexperienced against mistaking a mere fulness of the blood-vessels, arising from accidental causes, for a morbid congestion; and even against regarding the latter, which is often only of trivial importance, for an active state. Those who are not much acquainted with post-mortem appearances are too apt to regard much blood in the brain as a pathological condition, and this is a state very likely to be present in those cases requiring examination through an order from the coroner. For in the first place several days may have elapsed since death, and again, perhaps, the head only may have been opened; besides, if death has been sudden, which is very likely to have been the case, there is yet another reason for a fulness of blood, for there is more blood in the body of a person struck down in comparative health; so that if death be sudden, there will be an excess of it stagnating in the organs, of which excess from the body having been supine for some days, the brain has more than its share. Further, it is very important to remember that, if the head be examined first, the vessels are necessarily more full than if the body be previously opened, for in the latter case, the veins being cut and jugulars emptied, the blood flows out from the cerebral sinuses and surface. This difference may be observed constantly in the post-mortem room, according as the body or head is first examined. But if these accidents do not intrude, so that the head is opened after the body, and the person only dead a short time, even then, if the vessels of the dura mater, pia mater, and brain itself be very full of blood, all this is generally unimportant, and points merely to the cause of death by asphyxia. For in all pulmonary obstructions, whether primary or secondary, the brain is found congested like all the other organs.

There is no doubt that congestion of the brain is a real cause of serious symptoms and even of death. We do not deny the existence of congestion of the brain as a fatal disease: what we wish to say is that a fulness of the vessels after death cannot prove the occurrence of the primary congestion, and in fact post-mortem fulness of the vessels of the brain and its membranes is of no pathological value whatever as showing brain disease, because asphyxia produces it. On the other hand, anæmia of the brain does exclude congestion, and also proves that death was not through asphyxia, which always produces congestion. Our reason for saying all this so confidently, when even now we find writers in systems of medicine speaking of redness of the cerebral membranes as the great sign of inflammation, &c., is that we have seen all degrees of intensity of such congestion when there was no trace of cerebral disturbance during the life of the individual, and this we have seen not occasionally, but as a common occurrence. Again, it is not only the experience of morbid anatomists which declares intracranial congestion to be thus valueless. We will quote a passage from Kussmaul and Tenner's account of their elaborate researches on the effects of hæmorrhage as causing epilepsy. They say, "We could never deduce any results from the post-mortem examinations undertaken with a view to determine the state of fulness before death of the most important parts of the vascular system, viz. of the arteries and arterial capillaries, and even in the most favorable instance, when similar inquiries were directed towards the veins, our results could only be looked upon as approximate."

Thus, we see that it is only for lack of information that anyone would speak of congestion of the brain as a proof of a cause of death. We may infer reasonably from the circumstances of particular cases that death occurred from congestion of the brain, but we cannot prove it after death by inspection. We shall presently endeavour to show in what degree certain appearances of the brain are evidences of congestion.

**Air in the Subarachnoid Space.**—We speak of this because it is often met with, and students are constantly asking questions about it; and, moreover, we have seen it mentioned in a medical journal as a pathological condition. It arises simply from air rushing beneath the arachnoid when the calvaria is removed. It is not met with so often outside the hospital as here, where the opening of the head is more speedily effected than one usually sees in private. It will be observed that in sawing around the head the visceral arachnoid is slightly wounded, and then, when the lever is inserted under the bone, and the calvaria is suddenly pulled off, a vacuum is produced, and air rushes through the opening into the space beneath, and traverses the course of the blood-vessels, and thus arise the bubbles of air which have attracted notice.

**Œdema and Subarachnoid Effusion.**—Nothing is more common than to see the subarachnoid fluid increased in quantity, indeed, being the first thing observed on opening the skull, it often attracts more notice than it deserves; for, as a rule, it denotes merely a shrunken or wasted state of the brain accompanying a similar state of the other organs of the body, so that it is very common on our post-mortem table. In persons long ill with chronic wasting diseases the brain shrinks, the convolutions lose their plumpness, and separate. The visceral arachnoid still maintains its close apposition to the parietal; but the pia mater, fastened to the brain by its close vascular connection with it, follows the brain so that thus the subarachnoid space between the brain and arachnoid enlarges, and is filled with fluid. This we see bagging down in various places when the dura mater is removed, while the brain itself is generally pale. If the exudation were inflammatory it would be plastic or purulent, accompanied by congestion and other characters. Neither does the œdema accompany a dropsy of other parts, as we might imagine, for there is, indeed, no room for dropsical effusion unless the brain wastes; but, of course, when dropsy occurs elsewhere, there may be an increased wateriness of the brain and its membranes. It is important to know that this subarachnoid effusion is a mere passive condition, and denotes only simple atrophy and not an inflammatory process, for it often happens that we wish to know, by post-mortem examination, whether certain symptoms really arose from a diseased condition of the brain or not. Thus in a case of advanced tuberculosis or phthisis the patient has, towards its termination, delirium, or other brain symptoms, and we may suspect some disease in the brain similar to that in other parts; but upon examination we find the brain shrunken, with fluid on the surface, a condition denoting mere atrophy, thus showing there was no inflammation, for any active tubercular condition would be accompanied by effusion in the ventricles, and the brain would be swelled out and flattened against the skull, the convolutions being pressed together, so that there might be no fluid at all apparent.

**Chronic Thickening of the Pia-arachnoid; Pacchionian Bodies, &c.**—We mention these immediately after the account of excess of subarachnoid fluid we have just given because all these conditions constantly accompany each other, and evidently are closely connected. The chronic thickenings we are now considering always strike the inexperienced very forcibly, and often we have found it a great surprise to beginners that the thick, white, nodulated band along the course of the longitudinal fissure is not more spoken of at post-mortem examinations. Let us consider first the anatomy, and then shortly the signification, of this thickening.

In a typical case we find the proper membranes of an old person's brain to show a whitish opacity over the vertex, but we always see that the white opacity is greatest on the border of the longitudinal fissure for about an inch in breadth, then it diminishes outwards, so that, as we turn to the lateral aspect of the brain, the membranes are not remarkably thick, and at the base over the convolutions they are quite as thin as in early life. In an extreme example we see the thickening come down along the Sylvian fissure, and we also see patches on the posterior lobe corresponding to the points of entrance of the veins of the lower part of the cerebrum into the lateral sinus. Another point where we never fail to find the thickening is around the spot of exit of the venæ Galeni. Again, in some cases we see the membrane over the diamond-shaped space at the base of the brain thicker than natural; but when there is very marked thickening of this part of the membrane, our experience leads us to expect some former positive inflammation to appear in the history. Nevertheless it is common enough to find a slight thickening of the membranes at this spot, continuous with the thickening down the Sylvian fissure in the passive and senile states we are now speaking of.

Next we must notice that on this line of thickened membrane the so-called *Pacchionian bodies* are more numerous than usual, forming little white villous masses. If we look carefully at their position, we notice that they show an unquestionable relation to the points of exit of the cerebral veins that join the longitudinal sinus, being most developed around the more posterior of these veins, which slope more forwards in their course to join the sinus. In extreme cases we may find a tuft or two of these bodies where the small veins from the posterior lobe join the petrosal and lateral sinuses; if we observe them carefully, we find them to arise from the pia-arachnoid membrane at spots where its pia and arachnoid surfaces are blended. They are composed of a delicate fibrous tissue covered with epithelium, and are devoid of blood-vessels. At their origin they come in contact with the dura-arachnoid, which there bears a few of them on its surface also. They grow, soft as they are, through the tough dura mater, and penetrate, perhaps, into the longitudinal sinuses, and extending through the dura mater, they reach and excavate the skull, even pushing their way through this, so that they would perforate it; but usually the outer lamella rises a little above them, as seen in our specimens. They appear soon after birth and go on steadily increasing until, in old age, they reach a considerable size. They are sometimes found in a state of fatty degeneration or calcified, having in them sandy grains of "brainsand," so that they may be termed "psammoma." The sand grains are little balls of lime salts, separately encysted in fibre. We wish it to be noticed that there is a close relation between the development of Pacchionian bodies and the thickening of the membranes we have described. Again, there is a close relation between the same thickening and the quantity of subarachnoid fluid, so that when the membranes are thick the quantity of fluid is large, and *vice versâ.* Two other conditions accompany these changes. The bone thickens inwards, deepening the meningeal grooves, and the brain grows firmer while it wastes. This firmness of the brain, with the thickness and toughness of the membranes, renders it very easy to strip the membranes from the brain in elderly subjects. For the thick opaque membrane bears a hard pull without tearing, and the brain is separated from the surface already by the wider subarachnoid space. The reverse of the condition in young people makes separation of the membranes difficult in them.

So much for the anatomical appearances of these changes; now let us consider them in the endeavour to explain them. We first notice that the cerebral circulation is conducted under certain peculiar difficulties. Indeed, nature places certain difficulties in the way of a free circulation through the brain. We especially allude to the course of the upper cerebral veins, which is directed forwards, while the longitudinal sinus they enter runs backwards; the entering stream thus opposing the current it joins. Without attempting to give a detailed explanation of this, we see that, if the stream is feeble, this impediment to the exit of venous blood will prevent the brain becoming too easily emptied by gravitation in the erect posture, which would result in faintness. This opposition of the venous current is indeed, we believe, one of the most important means of accommodation of our species to the erect posture, though we do not think it has yet received any notice in that light. But while there is thus an advantage during feebler states of the blood-stream, the impediment caused by the opposition of the current is a serious source of congestion in states of active pressure of circulation, so that the hinder cerebral veins, which enter most obliquely, and have the greatest backward rush in the sinus to resist, are greatly impeded in the discharge of their blood, and are thus held in a state of congestion. Now, we know that the stream is strongest in the centre of a vessel, and weakest at its side, and hence we see that the lateral part of the obstructed stream will feel the impediment most seriously; but this lateral part is that which receives the last venules from the membrane around the exit, and thus it happens that the effect of the stasis induced by the obliquity of the veins is found greatest at this part. The effects of such congestion we are familiar with in heart disease, &c.; they are summed up thus:—An overgrowth of fibrous tissue, and a loss of the higher functioning elements, with a constriction of blood-vessels by the excess of fibre, so that in the neighbourhood of the exit of the veins we find a great thickening of the membranes; while at their very mouth, where the congestion is at its extreme, we see papillose outgrowths from the arachnoid, such as we occasionally see on the surface of the liver in cases of very protracted and severe cardiac congestion. These papillose outgrowths are the *Pacchionian bodies.*

The same chronic thickening of the connective substance, with wasting of the functioning part of the organ, similar to what occurs in the liver or old cardiac obstructions, will explain how it is that the brain grows harder and firmer. It undergoes, indeed, a slight general sclerosis through the impediment we are describing. The wasting of the brain induces a complementary excess of subarachnoid fluid, whose part it is to fill the space from which the brain recedes; it appears also that the shrinking brain keeps a sort of suction upon the dura mater, tending to fill its vessels, and so induce thickening of the inner surface of the skull, but whether this be the means or not, there is constantly an inward thickening of the skull, serving to reduce its cavity in some proportion to the decrease of the brain.

**Meningitis, or Inflammation of the Pia-arachnoid—Leptomeningitis.**—It is in connection with the pia-arachnoid and the subarachnoid space that the products are formed in inflammation of the membranes of the brain, and thus it is under this head that some of the most important acute diseases of the brain are to be noticed. Dura-arachnitis, with its effusion between the arachnoid surfaces, is an affection, as we have said, intimately connected with disease of dura mater, and arises from some external cause; whilst the effusion beneath the arachnoid, and immediately in connection with the cerebral substance, has its cause within, and constitutes the idiopathic inflammation of, the membranes; with this, however, there is constantly a very slight exudation on the free serous surface; but this is insignificant compared with the purulent arachnitis in the same part, arising from injury, &c., of which we before spoke. In ordinary idiopathic meningitis, the effusion, be it lymph or pus, is poured out from the pia mater; a structure which, as you know, consists of a most rich plexus of vessels, held together by delicate areolar tissue, following all the inequalities of the convolutions, and lying beneath the level surface of the visceral arachnoid membrane, with which it is connected by fine filaments, forming a delicate texture that crosses the subarachnoid space. When the pia mater is affected there can be no doubt that the adjacent brain structure is also involved, and the ventricles also suffer, for into them the choroid plexus of the pia mater projects, carrying with it the inflammation; as, however, the changes are less appreciable here than in the membranes, we are accustomed to speak of the affection as meningitis, although perhaps in many cases the whole cerebral structure is involved.

Acute meningitis or, more properly speaking, pia-arachnitis, is mainly of two kinds, *simple* and *tubercular*, the latter being also known as acute hydrocephalus. It is highly important to distinguish between these two forms, which present characteristic differences.

**Simple Meningitis—Pia-arachnitis.**—In this form of disease we find the surface of the brain, in the space beneath the visceral arachnoid, covered with lymph or purulent lymph of a greenish colour. This is especially seen in the sulci between the convolutions, but may exist in such great quantity that the cineritious structure is quite hidden; and thus, directly we remove the dura mater, we are struck with seeing this inflammatory effusion completely covering both hemispheres, as if a layer of green purulent lymph had been poured over them; this appearance is well seen in our drawings. Placing the finger on the brain, we perceive at once that the effusion is beneath the arachnoid, and cannot be removed without cutting this membrane. On scraping the external surface, it is likely that a small quantity of exudation may be seen on the scalpel, for it is scarcely possible for such an acute inflammation to exist on one side of a membrane without the other being slightly affected, yet on microscopic examination of the matters removed by scraping, we have often found only epithelium and detritus. On removing the brain we find the sides covered in the same manner, but to a less degree; and on looking at the base, there is generally but little effusion there. The ventricles are not necessarily, we think, affected; but generally the fluid in them is increased, and is somewhat turbid. On attempting to remove the pia mater from the surface of the convolutions, the cineritious structure is found to stick to it, and to tear when any force is used, showing that it has also been involved; and, indeed, it will be impossible to say in such cases what parts of the brain have not suffered, though it is only on the free surfaces that the changes are markedly perceptible. In one of the last cases of this kind, which we examined, the sinuses of the dura mater were filled with firm coagula. It must be remembered that no tubercles are found in this disease, either in the brain or any other part of the body; indeed its pathology is scarcely known, though, in all probability, it occurs in the exanthemata; for example, as a sequence of scarlatina. It is a very acute disease, running its course in three or four days. Not many months ago we examined a child with this form of meningitis, who had been ill only three days: every part of the body except the brain was healthy. It is possible that it may originate in injury, in the same way as the dura-arachnitis of which we spoke; and under these circumstances one hemisphere may be involved and not the other. We have seen two or three instances where an inflammation of the spinal cord has extended up to the brain, and produced a meningitis, with the greatest amount of lymph at the base. These cases have closely resembled the epidemic cerebro-spinal meningitis.

**Tubercular Meningitis, or Acute Hydrocephalus.**—This is a form of disease quite peculiar, and easy to recognise; for, apart from the presence of tubercles, the characters of the inflammation are such that we may know at once what is the kind of affection we have before us. The disease is less acute than that last mentioned, generally running a course of two or three weeks, with symptoms less violent. On removing the dura mater, we see the surface of the brain flattened, owing to the pressure which has been exerted by the ventricular fluid within forcing the hemispheres against the skull. The convolutions are thus pressed together, and the sulci almost obliterated, so that the brain may look like an even mass, with vessels ramifying over it. The arachnoid will have somewhat lost its lustre, but on scraping it very little indeed can be obtained on the scalpel. Beneath the arachnoid no lymph is seen, unless perhaps, on looking at the sides, a small quantity may be observed in one or two sulci; thus the surface of the brain presents no marked inflammatory products, as in the acute and simple form of arachnitis just described. On removing the brain very distinctive appearances are seen, chiefly in the presence of a little tenacious lymph at the base, in the diamond-shaped space, and about the pons Varolii, and cerebellum, often indeed also surrounding and involving the cranial nerves. The collection of lymph in these parts is so characteristic that German pathologists call this disease "meningitis of the base." If this lymph be examined, some tubercles may be found in it attached to the small vessels that cross the space. Similar exudation is also generally seen proceeding upwards in the fissure of Sylvius, so that, if the lobes be separated, a quantity of it may be found connecting them together. The true miliary tubercles are not generally seen from the surface of the brain, but must be searched for in the pia mater after its separation. They are readily brought to view by stripping off the visceral arachnoid, together with the pia mater, and carefully examining the portions of the latter membrane which dip down between the convolutions; it is on these especially that we shall perceive the tubercles. It is best to place the membrane in water over a dark surface, when the tubercles readily appear as white dots. Probably tubercles sometimes exist before the inflammatory exudation occurs, but during the inflammation a still further and more rapid production of them takes place, in the same way as in the phthisical lung and other tuberculous organs. We are under the impression also that the simple miliary tubercles are not of long duration before they show signs of their presence. We think this because in many children who have died of general tuberculosis there have been no cerebral symptoms; and when we have carefully examined the membranes to see if, perchance, any tubercles might be present, we have hitherto always failed to find them; but we have met with some facts which prove that the tubercles may be produced before the inflammation. Thus, we have found some of the tubercles so large, and so altered by cheesy changes, that they must have been older than the inflammation of two weeks' duration; and we once had a case of phthisis where a man had been quite sensible to the time of his death, and yet in the membranes of the brain were many conspicuous tubercles, some of them large and caseous, like those we have just now alluded to. It thus appears that sometimes coarse miliary tubercles precede the general tubercular inflammation, in which the crowds of minute tubercles appear, though this circumstance is only occasional. And now as regards the ventricles. On slicing the brain until they are reached, we find the roof bulging out, and the corpus callosum lifted and carried backwards, owing to the quantity of fluid present, which reaches sometimes to as much as four ounces, though it is generally less; the fluid is itself more opaque than in health, and becomes somewhat more so on boiling, and we believe the specific gravity is increased, this having been 1010 in some cases which we have examined. The circumference of the ventricle is soft, sometimes so exceedingly soft as to be quite diffluent; and this is more the case in the central parts than at the sides; thus the fornix, septum lucidum, &c., are sometimes quite broken down, shreds of the tissue hanging into the ventricles. If the septum be not broken, the foramen of Monro between the two cavities is found enlarged. Much controversy formerly existed as to the cause of this softening, whether it be inflammatory, or due merely to the ventricular effusion; but there is no doubt it is an inflammatory œdema, for the microscope reveals inflammatory products, and a mere soddening of the cerebral tissue will fail to produce a like effect. The microscope does not show, it is true, those granule masses so characteristic of subinflammatory states, but it shows the elements of these, or the fatty granules collected along the course of the capillary vessels, to explain which a passive softening is not sufficient. A cause of local softening may sometimes be found in an actual obstruction of the vessels by a mass of tubercle. This may even be productive of small hæmorrhages, as seen in connection with tubercle in other organs. If the surface of the ventricles be carefully examined, it will sometimes be found granular. This may be overlooked, unless the brain be held to the light, but then the surface appears as if it had had some fine sand sprinkled over it; this condition may continue downwards into the fourth ventricle. As we said, when speaking of simple meningitis, we are apt to regard these affections as inflammation of the membranes only; but, in all probability, the brain structure is more or less involved, though the evidence of its disease is less appreciable. The superficial part of the brain can hardly avoid suffering when the pia mater is affected; but its implication is demonstrated, even to the naked eye, by the softness and adhesiveness of the cineritious structure. Thus, in attempting to remove the membranes, the grey matter comes off with them and adheres to them, and tears up in its layers, which may be thus separated from one another.

The microscope will make quite clear the degree and manner in which the brain is implicated. If, in the first place, we strip a little piece of the membrane from a convolution in the Sylvian fissure and examine it by a low power, we see that the tubercles follow the course of the small arteries. If then we choose a favorable spot, and apply a higher power, we see that the tubercles, which are composed of well-formed lymphoid corpuscles, are enclosed between the vessel and its perivascular sheath, often compressing the vessel and narrowing it. If now we harden a portion of the brain with the membrane attached, and cut sections through both brain and membrane, we see that the tubercles, still within the sheath, follow the vessels, as they enter into the cortex of the brain. Thus one understands how the functions of the brain should show such severe derangement in tubercular meningitis. In no other part of the body are tubercles seen more clearly limiting themselves to the course of the blood-vessels. This relation has given rise to several hypotheses; some observers think there is a multiplication of cells that form an endothelium to the perivascular sheath, others prefer a view that supposes the corpuscles of the outer coat of the small artery to multiply. Others uphold the theory of outwandering of white cells of the blood into the sheath. The difficulty of arriving at the exact facts of the case are very great, although it is evident that the tubercles certainly are close around the small arteries, and the appearances admit of all these explanations.

Tubercular meningitis may be found not only in connection with tubercle elsewhere, but occasionally in association with scrofulous glands and chronic abscesses; thus it is not uncommon for a fatal tubercular meningitis to follow on a long-standing caseous degeneration of the bronchial glands apparently arising in connection with the bronchitis of measles. Such cases suggest an infection from these glands. We should add that true tubercle bacilli have been found in tubercular meningitis. In the same way a source of infection may exist in the internal ear. This gives rise to a great difficulty in diagnosis.

To recapitulate: the distinctive characters of tubercular meningitis are, the absence of lymph on the surface of the brain (in this it differs from simple meningitis), a flattening of the hemispheres, lymph at the base, tubercles in the pia mater following the course of the vessels, and increased fluid in the ventricles, with softening of their walls. Even if tubercles were not visible to the naked eye, the rest of these characters would at once indicate the nature of the disease, and the microscope will always find the tubercles along the vessels.

**Non-tubercular Basic Meningitis.**—Although we have spoken of tubercular meningitis as being mostly basic, yet the contrary is not invariably true that basic meningitis is always tubercular. It may be, however, often of this nature when no tubercles are found in the pia mater whilst they abundantly exist in the lungs and elsewhere. But besides these cases we sometimes meet with inflammation confined to the base of the brain both in infants and adults. In the former the age is generally much younger than in the tubercular cases, and the illness of but few days' duration. We sometimes meet with cases of longer duration and accompanied by large effusion in the ventricles. These are probably the kind of cases which occasionally recover. If the recovery is not complete, certain symptoms remain showing that the brain has been permanently damaged.

We know the case of a little girl, now ten years of age, who had severe cerebral symptoms which were regarded as those of inflammation of the brain some years before. When she recovered she was found to be perfectly helpless both in body and mind, and has remained in an idiotic condition ever since. In two cases of children which were regarded as meningitis the patients recovered, but were found to be stone deaf, as they have been ever since. The meningitis at the base may have involved the auditory nerves, and the inflammation run along them to the internal ear. Sometimes as a result of the acute inflammation the head enlarges and remains permanently of great size. In the case of a man who died three years after a severe injury to the head followed by cerebral symptoms a pint of fluid was found in the ventricles.

**Syphilitic Meningitis.**—We have occasionally, though rarely, met with a primary pia-arachnitis in cases of decided constitutional syphilis. These cases resemble tubercular meningitis more than the acute simple form. But there was more lymph effused, and the tubercle granulations were absent. In neither of the cases was there a syphilitic affection of the dura mater. The disease was acute. Lancereaux mentions a few such cases, and we are convinced that syphilis is an occasional cause of acute meningitis.

**Hæmorrhage beneath the Arachnoid.**—This occurs under the same circumstances as bleeding between the arachnoid membranes already mentioned. The blood may run beneath the arachnoid when the brain is injured, or when meningeal apoplexy is a spontaneous disease, or when an apoplexy reaches the surface, or an aneurysm bursts, or a tumour bleeds there. Sometimes meningeal apoplexy occurs as a fatal result of purpura or scurvy. It is not uncommon, too, to find blood beneath the arachnoid, at the base of the brain, when blood bursts into the lateral ventricles, and then passes by the third to the fourth ventricle, and from thence into the subarachnoid space of the medulla oblongata, pons Varolii, and adjacent parts. As a spontaneous apoplexy it is said to be almost peculiar to advanced life, but we have met with it in a few cases in young persons, as already mentioned under dura mater. The remains of old sanguineous apoplexies may be occasionally found on the convolutions, especially near the fissure of Sylvius, whence often the blood has issued.

**Tumours of the Pia-arachnoid Membrane.**—*Secondary* tumours here, as elsewhere, have the same character as the primary growths from which they arise. We have met with carcinoma, sarcoma, and lymphoma; probably every kind of malignant tumour may occur here. Thus pigmented sarcoma or melanosarcoma has not infrequently been found widely diffused in the cerebral and spinal pia mater. These tumours invade the cerebral tissue, and are secondary to melanosarcoma of the skin and elsewhere. We have twice known epithelial carcinoma from the face penetrate to the membranes and the brain; other cancers from the bones will do the same.

But chiefly deserving attention here are the *primary* tumours of the membranes. As fibrous structures these membranes give rise to *fibroma* and *sarcoma*, and this we should expect on the general principle that tumours have a resemblance to their matrix. The sarcomatous growths are harder or softer; they grow inwards into the brain. Giant or *myeloid* cells may be found in them; there may be hæmorrhage into their texture. Virchow has give the name *melanoma* to very rare tumours that arise as an increase of the natural pigment which we find in the pia mater, especially over the medulla oblongata. In two cases this pigment has been found to take the form of numerous small tumours along the whole membrane; in Rokitansky's case this went with similar pigment growth in the skin; these do not invade the cerebral tissue, and are primary. Occasionally we meet with *angioma* or erectile tumour. One such case is given in the 'Path. Trans.' by Mr. Morris. A very interesting form of growth is villous tumour (*papilloma*) of the pia mater; we have twice met with this about the pons. Another interesting tumour is *cholesteatoma*, a specimen of which may be seen on our shelves. The man from whom it came was in the Hospital suffering from cerebral symptoms, and left here to go to Colney Hatch Lunatic Asylum, where he died; this tumour, of about the size of an egg, was found at the base of the brain, loosely connected with the arachnoid, and was sent to us by Mr. Tyerman. It was of pearly lustre, and broke to pieces when handled, just as the matter from a sebaceous tumour, with which, indeed, it is in all respects identical, for it consists of a mass of cells containing fatty matter, mixed with plates of cholesterine, and hence the name.

We have met with a hard cartilage-like growth extending from the membranes into the cerebellum. Its structure was like hyaline *enchondroma* in parts, and in parts like osteoid cartilage. It has some resemblance to a curious *waxy* tumour shown by Dr. Murchison at the Pathological Society ('Trans.,' vol. xiii). We have also met with *epithelioma* of the pia-arachnoid. Occasionally *dermoid cysts* are met with. These are considered by Mr. Bland Sutton to be fœtal relics.

Although much less subject to this than the dura-arachnoid the pia-arachnoid sometimes shows small plates of true bone in its substance, as in one specimen over the frontal lobes. We shall find that the arachnoid of the cord is curiously subject to osteoid plates. Some instances of small *lipomas* in the meninges have occurred, generally about the regions of the brain corresponding to the basi-sphenoid, or on the corpus callosum. One of the size of a bean upon the auditory nerve is said to have produced deafness.

We have twice met with the small soft cellular tumours under the pons which have been thought to arise from a persistence of the fœtal notochord, the structure of which they resemble, so that Virchow calls them *chordoma.*

## VENTRICLES

**Inflammation.**—*Acute.*—This, in its commonest form, we just now spoke of under "Acute Hydrocephalus." The fact of the ventricles so suffering has given this synonym to tubercular meningitis. Acute inflammation of the ventricles is rarely found except in general meningitis, but we have known cases where pyæmic abscesses have burst into the ventricles and set up a foul and general inflammation within. Dr. Delafield, of New York, describes an example of general idiopathic inflammation of the lining of the ventricles. We have in a few cases seen suppuration within the ventricles as a consequence of injury to the head, and in one case a distinct lining membrane of lymph. One of the most marked cases of the kind occurred lately in the Hospital. It was that of a child whose head began to enlarge soon after birth, and who also was the subject of spina bifida. The latter was tapped and injected, and the child died seven weeks afterwards. The brain was in a pulp, and the ventricles contained a thick viscid purulent fluid. The interior of these cavities had shreds of membrane hanging from it. The technical name for such a condition would be *pyocephalus.* It was very remarkable also to find, in this infant, a closely adherent pericardium.

*Chronic.*—Chronic hydrocephalus has a somewhat complex pathology, and probably more than one disease is included under this name, Common as the disease is, yet, being of long duration, the chances are few of our being able to watch a case to its termination, and to examine the parts after death. Those who now and then have had an opportunity of so doing have reported nothing but increased ventricular effusion, which sufficiently shows that the morbid appearances are not very great; and this has led some to suppose that the disease is in every respect analogous to those examples of pleuritic effusion, where we suppose the membrane to be in a morbid state from the fact of the increased secretion, although there are no changes appreciable to the naked eye. It has also been thought that the effusion might be a dropsy produced by some local inflammatory process, whereby the veins of Galen are impeded. It is probable, however, that further researches may show that the disease owns a variety of causes, for it occurs under such diverse circumstances. Thus, there is the hydrocephalus with which some infants are born; then the more common variety, which comes on some weeks after birth; generally within the first three months, and there is also another, in grown-up persons, of which we have now seen many examples.

The history of acute hydrocephalus shows that the ventricles, through the intervention of the *velum interpositum*, may become the seat of an active effusion. Sometimes on examining the communication between the fourth ventricle and the subarachnoid space we find an obstruction of this opening; that this was one of the causes of effusion into the ventricles was held by Mr. Hilton, with whom we had an opportunity of seeing a case of the kind. A gentleman, who had never since birth been in full possession of his mental faculties, died rather suddenly, and an immense ventricular effusion was found in the brain. The arachnoid appeared everywhere opaque, particularly at the base, and it seemed to close the fourth ventricle. Since this, we have had other cases of the same kind, that is of persons with large heads and somewhat feeble minded, in which there was reason to suppose that ventricular effusion had occurred in childhood. Thus, a gentleman of fifty, with a head notoriously large, and feeble in mind, died rather suddenly, and his ventricles were found very large and distended with fluid. A few years ago a schoolmaster was put on his trial for causing the death of a boy from flogging. He was a dull, stupid boy with a large head. The post-mortem showed a large quantity of fluid in the ventricles. One of the most remarkable cases of hydrocephalus recorded was that of Cardinal, whose skull is in the Museum, and whose ventricles contained a gallon of fluid. This had burst through the corpus callosum and forced the brain to the base of the skull. In a case of chronic hydrocephalus in a child two years old, described by Dr. Baxter, where he found five pints of fluid in the lateral ventricles, the fourth ventricle was closed as Hilton had described. Dr. Baxter had paid much attention to the point, but had failed to find the closure in any case except this one.

Dr. Goodhart has put up in the Museum sections of the fourth ventricle, and he states that in such cases there is evidence of an old inflammation about the foramen magnum, but he has never made out a single closure of the fourth ventricle.

In one of our cases the brain was adherent firmly around the foramen magnum, and the membranes and choroid plexus at the infundibular opening of the fourth ventricle were very thick, almost closing the opening. But the most remarkable case we have seen was one in which on opening the skull there appeared a large bladder like a hydatid separating the left cerebral convolutions. This proved to be the immensely dilated lateral ventricle which had thrust its way through to the surface, where it occupied about two superficial inches. The cause of this was found to be a papillose tumour in the floor of the fourth ventricle composed of ependymal structure. It was of the size of a small hazel-nut, and completely cut off the cavity of the ventricle. Two very similar cases are given by Virchow. Again, we have known the ventricles of the brain distended much in a case of pressure on the spinal canal through disease of the cervical spine; thus cerebral symptoms became added to the spinal. Another certain cause of expansion of the ventricles is tumour or tubercle under the *tentorium cerebelli*; whether it grow in the pons or the cerebellum, it compresses the fourth ventricle, and prevents the free escape of the cerebro-spinal fluid. In explaining these results we must remember that this fluid is secreted into the ventricle by the choroid plexuses, and finds its way from them down through the opening of the fourth ventricle into the subarachnoid space. It might be mentioned that the fourth ventricle is sometimes found extremely dilated as well as the lateral ventricles in hydrocephalus.

As we said before, in chronic hydrocephalus the fluid is always within the ventricles; an external hydrocephalus is spoken of, but we never saw it.

A chronic inflammation of the ventricles, shown by a granular condition of their lining membrane or ependyma is sometimes met with. We have already mentioned such a condition in the brain in acute hydrocephalus; but it may be constantly met with in the ventricles of cases where the meninges are thickened. In some such the whole of the interior was covered with large translucent granulations, which once we saw as large as hemp-seeds, giving the surface the appearance of the leaf of an ice-plant. In other cases the change is more local. The substance of these little excrescences is much the same as that of the ependyma, of which they represent warty or papillose states. If we look carefully we nearly constantly find a few such grains on the *septum lucidum* about the entrance of the vein of this septum into the choroid plexus; these are evidently equivalent to internal Pacchionian bodies.

**Blood in the Ventricles.**—Blood is found in the ventricles in some cases of apoplexy. The entrance of blood into the ventricles is a grave occurrence in the course of an apoplexy. The blood will sometimes fill the whole of the ventricular system and coagulate there, so that if we remove it carefully under water a mould of the ventricles is found to have been taken most exactly by it. It extends down the subarachnoid space of the cord, though it is not usually followed there, and this part of its history is overlooked.

In cases of severe injury to the head the ventricular liquid is usually bloody. This we generally find to arise from rupture of the *septum lucidum*. It is at first surprising that this part of the brain, which is protected by its central position and placed in a water-bed, should suffer more than others; yet often it suffers disproportionately. Thus we have frequently seen it torn rather freely when little injury was done to the other parts, and once it was ecchymosed after a blow when there was no fracture of the skull. But although the bath of fluid it lies in is very admirably suited to reduce slight vibrations on their way to the septum, yet the water will move *en masse* against the septum when the head is forcibly thrown against an obstacle, if the slightest alteration in the shape of the skull be made in the supreme moment of contact, and thus it becomes very liable to burst through, as we may often find it, or seriously torn, as is more frequent.

**Closure of Cornua.**—The posterior cornu of one or both lateral ventricles is often found closed up, as Esquirol long ago remarked. This is caused by the inward pressure of a deep sulcus of the surface. Sometimes the communication of the cornu with the ventricle is cut off so that the cornu forms a cyst. This occurrence has no pathological significance. Grohe gives an account of three large cysts in connection with a sarcomatous tumour of the brain. He believed these to arise by alteration of the lateral ventricle.

**Tumours in the Ventricles.**—We have mentioned warty growths from the ependyma in the fourth ventricle, and we have met with a tumour the size of a walnut and a papilloma. The ventricular surfaces are, however, very little prone to originate either morbid growths or inflammation.

In cases of chronic atrophy of the brain, and in old people, it is not uncommon to find *cystic* formations in the *choroid plexus*. The cysts may be two or three in number, and of the size of peas, or larger. They were formerly called hydatids. Changes may occur in these, converting them to *earthy concretions ;* these then are found as rounded bodies composed of carbonate and phosphate of lime, and have no analogy to bone.

*Hydatids* occasionally occur ; we have a preparation showing two cysticerci which came from the lateral ventricles.

Dr. Cayley showed at the Pathological Society the brain of an infant which had a cyst the size of a duck's egg, springing from the choroid plexus and filling the ventricle. It had forced its way through the corpus callosum to the surface of the brain. It had thin walls and contained clear serum.

## CEREBRUM

**Hypertrophy.**—We have no special experience of this condition ; it is very questionable whether true hypertrophy of the brain exists. We all know that scrofulous children often have large heads ; but this unusual size of the head is generally only apparent, being due to the smallness of their limbs, and it does not signify enlarged brain. In cases which have been called hypertrophy this condition was supposed to be the cause of death. Thus, occasionally we hear of a child who has had cerebral symptoms, so that it was expected that hydrocephalic disease would be found ; but upon removing the calvaria the hemispheres were seen to be flattened against the skull, or actually protruding through removal of the pressure, just as if there was a large ventricular effusion; but on examining the central parts of the brain no fluid was found. On the contrary, the ventricles were small, and, therefore, the size was considered due to an actual growth of the cerebral structure, or a morbid hypertrophy. We have ourselves no experience of this state. In cases that have occurred lately the hypertrophy was found to be due to increase of the neurilemma or cement between the fibres. Dr. Fagge, in his work on 'Medicine,' relates two cases of hypertrophy on the authority of Dr. Beach.

**Atrophy.**—This may be general or local. A *general atrophy* or atrophic induration accompanies and causes the subarachnoid effusion we have recently described. Thus, in old age, in persons long bedridden by sickness; in drunkenness, lead poisoning, &c., and in old cases of mania, the brain is found shrunken, not filling the skull, the convolutions parted asunder and pale. As to the brain itself, its atrophy is seen in the following particulars. The organ is shrunken, as already described; its ventricles are commonly dilated, their lining thick and often granular on its surface; the surface of the thalamus and, in a less degree, of the corpus striatum, is sinuous and shrivelled looking. But it is not only the surface that shows the change, for everywhere throughout the brain we notice its tissue shrinking away from the blood-vessels, leaving them in wide canals twice or more their own width. This is most conspicuous about the perforated spots, *i. e.* in the lower part of the corpus striatum. The microscope shows in such brains, especially in the thickened ependyma, but also in the neuroglia throughout the organ, the presence of the so-called amyloid or colloid corpuscles, small round bodies of laminated structure like to starch grains. Often, but not always, these are stained purple by iodine, sometimes they are tinted brown, sometimes not at all specially coloured by it. They have always excited great interest through their close resemblance to vegetable starch in their more perfect forms,—sometimes they even have the hilus, just like starch grains.

The most marked changes have been found in *general paralysis of the insane*, the degenerations being seen in all the structures, including membranes, brain tissue, and blood-vessels, suggesting a change somewhat analogous to what is seen in the granular atrophic kidney. The membranes may be adherent and thickened; sometimes old extravasations of blood are found presenting the appearances already described; the vessels too may have undergone a morbid calcareous change. These degenerative changes in the brain are in some cases of a simple atrophic character, in others of a kind to which the term chronic inflammation is usually applied.

In a case lately examined the membranes were thick, and there was a nuclear growth on their surface and adjacent tissues, so that the term meningo-cerebritis might be very rightly applied. In cases, however, where the naked-eye appearances are not well marked, it has been shown by Bevan Lewis, who has paid great attention to the subject, that the blood-vessels are distended, and the perivascular sheath obstructed by leucocytes and pigmented crystals; that the grey cells have undergone a gradual degeneration and become darker, sometimes have lost their processes or become fibrous, so that in a very remarkable manner the fibres are seen passing through the cell. The axis-cylinders may for a long time remain intact. The cells may sometimes show not only a fuscous or pigmentary degeneration, but lose their nucleus and undergo a vacuolation. It is a question how far certain changes are primary or how far secondary, for since secondary degenerations have been studied it is evident that the nutrition of certain nerve structures depends upon the integrity of very distant parts.

Although general paralysis may come on at an early period of life it is difficult to say in what respect the morbid changes found in the brains differ from those which naturally occur in old age. In senile decay, or in *senile dementia*, accompanied by weakness both of body and mind, we often find the membranes thickened, the Pacchionian bodies large, the convolutions shrunken with the sulci correspondingly deep, the vessels thickened, the ventricles containing increased fluid, the nerve-cells undergoing fatty or pigmentary degeneration, and the nerve-fibres much wasted; and with these, perhaps, some increase of connective tissue. In old cases of insanity both Westphal and Ringrose Atkins have found marked atrophy and degeneration of the spinal cord accompanying similar changes in the brain.

*Local wasting* arising from diseased blood-vessels, inflammatory softening, &c., is very common. Where the cause is not very evident, we are obliged to be content with the term atrophy. Such cases are occasionally met with where a few convolutions have disappeared, or a yet larger part of the brain may be missing.

In some cases of congenital paralysis, where from birth one side of the body has been atrophied and useless, the opposite side of the brain has been found wasted. This has, apparently, resulted from disease in fœtal life. In one case of a child who was born hemiplegic, and died soon after, a clot of blood was found on one side of the brain. This the doctor very naturally attributed to a blow which the mother had received on the lower part of her abdomen during her pregnancy. If, then, the child had grown up, an atrophy of this part of the brain would have ensued, with a permanent paralysis of the other side of the body. Several cases have been recorded of meningeal hæmorrhage in infants, which are supposed to have occurred from difficult labours, the effects of which, if not immediately fatal, might be shown by a hemiplegia. In other cases there has been either a history of effusion of blood, or other affection of the brain, at a later period of childhood. Of this kind Dr. Taylor described an interesting case in our Reports. A young man, aged twenty-five, had had from childhood a wasted and weak left arm and leg, arising probably from an apoplectic attack when he was five years old. The central ganglia on the right side were smaller than those on the left, and there was a corresponding diminution in size of the right crus, medulla, and anterior pyramid; but the left lobe of the cerebellum was much smaller than the right, according to the rule of the cerebellum being affected on the opposite side to that of the cerebrum in similar forms of disease; the wasting then continued down the left lateral column.

In a similar case of Schroeder van der Kolk's, a girl, paralysed from infancy on the right side, had the left hemisphere and ganglia smaller than those on the opposite side, as well as crus, pons, and pyramids; but the right lobe of the cerebellum was the part wasted in this organ. It has been thought by some that a hemiplegia may occur in children of the same nature as infantile paralysis; that is, a polio-myelitis affecting the motor tract within the cranium.

Further observations are required to show the more partial wasting of the motor tract in local paralyses and congenital malformations, but Gowers described the case of a man with congenital absence of left hand in whose brain he found the middle portion of the right ascending parietal convolution half the size of its fellow. In another case of the kind the nerves of the cervical enlargement on one side were smaller than those of the other, as well as the cord itself.

In several cases of amputated limbs the grey matter of the cord, whence the nerves to the limb proceed, has been found atrophied; probably from disuse. When we speak of degeneration of the cord we shall again allude to its association with similar processes in the brain. In experiments on animals, the results of which are confirmed to a certain extent by cases of disease in the human subject, degeneration of the motor tract, in connection with that of the spinal cord, has been found commencing immediately beneath the convolutions in which the lesions had taken place, and then proceeding downwards along the fibres of the corona radiata to the internal capsule, *i. e.* the white band separating the lenticular ganglion from the caudate and thalamus; its middle and anterior portion containing the motor fibres being more particularly affected. The degeneration then passes on to the cord by the lower layer of the crus cerebri. The course of the degeneration downwards from operations on the cortex may be found described by Dr. Ferrier, and it was well seen in the case of the dog brought to the International Medical Congress by Dr. Goltz. The course of degeneration along the ascending sensory fibres of the brain has not yet been made out.

*Cystic degenerations.*—These may be of various kinds: there are the *apoplectic cysts* already described, and there are others whose origin is obscure, but which are often called *inflammatory cysts*, as they are found in connection with evidence of inflammation of the brain or membranes. There are also cysts of various shapes and sizes sometimes found in the anterior lobe, sometimes in the posterior lobe, or occasionally in the cerebellum. In the Museum is the brain of an old man containing a large cyst in the right anterior lobe, and communicating with the ventricles by a smooth opening. Then there are cysts which are sometimes found in the brain of infants, or rather clefts running from the surface to the ventricle, and which may be congenital: these have been styled *parencephalic*.

Besides these there is the remarkable cystic disease of the brain which has been studied by Dr. Savage and Dr. Hale White. The brain is full of holes, which are round and smooth within, giving it the appearance of Gruyère cheese, after which it has been named. They look as if pieces of tissue had been punched out; some of the smaller cysts, when carefully examined, appeared to be formed of dilatations of the perivascular canals, but whether this is the origin of the larger ones is questionable. It is remarkable that some of these cases occurred in connection with cystic disease of the liver and kidney. Now, as regards the kidney, it seems true that the cysts may be formed of dilated ducts; but in the liver they seem rather due to evacuolation of the hepatic cells. It may happen, therefore, that some similar evacuolation of the white matter may occur in the brain, and so produce the cysts. The cystic disease sometimes extends to the pons, medulla, and cerebellum.

**Injury.**—Injuries to the brain are of a dangerous character; yet if no general inflammatory action ensue, a considerable loss of substance may occur without destroying life. A case is reported of recovery after the escape of brain tissue through the external ear in fracture of the base, and several where large portions of the frontal lobe have come away. An incised wound may heal, as in any other organ, and, without doubt, slight bruises to the substance are constantly recovered from. We have already given an account of two cases where there was evidence of recovery from severe bruising of the brain; such cases would clinically be called concussion. No doubt in most cases of concussion such bruising is the cause of the symptoms. We have not unfrequently had opportunities of seeing brains which have suffered the slighter degrees of concussion; thus, sometimes when death has occurred from other injuries the cineritious structure is found bruised and ecchymosed, although few symptoms were observed during life. In more severe cases of injury to the head, with or without fracture to the skull, such an ecchymosed condition is always found.

The relation of the bruised part of the brain to the part of the skull that received the blow is of great importance. We will shortly state the law of it. In blows of great velocity and little weight the skull is broken, and the brain bruised, immediately under the injury in the bone. But in blows of low velocity and heavy weight, as especially in falls, while the skull is fractured at the point struck, the brain is always found bruised at a point opposite to this; so that if a man falls on the back of his head, rather to one side, as is usual, the skull is broken at the back, and the blood outside the dura mater is always just where the fracture is; but the front of the brain on the other side is the part bruised. The membranes are here usually torn, and we find the blood effused in the arachnoid and subarachnoid spaces. The surface of the brain, to a greater or less depth, is bruised, and mixed with blood, while about the bruised part are some conspicuous round extravasations. The bruising affects the grey matter more than the white, even where by the course of the sulci both are removed equally from the surface. It is easy to understand why the brain should be bruised under the spot struck in the sharp light blow; but it is not so easy to see why a heavy blow should smash the brain at a point opposite the seat of its impact, when the skull is always broken at the point struck, while the brain comparatively escapes there. This fact is expressed by the term *contre-coup*. The common explanation supposes that the vibrations in the skull, which radiate from the impact, pass round the sphere of bone, and, converging at the opposite point, are not able to fracture the skull again there (*contre-coup* of the bone is very rare), so they expend their fury on the soft parts, tearing up the brain. But in using this explanation we must remember that the skull is not a sphere, but its bone is rough and has inward projections; again, its base fits at certain parts very close indeed to the brain; such parts being the roof of the orbit and the depth of the middle fossa; and if we see a large number of cases of *contre-coup* we shall be surprised to find how constantly those parts of the brain which touch the base of the skull at the points named are by far most severely lacerated. The reason of this, no doubt, is that when the form of the skull is slightly changed by its yielding in the supreme moment of impact, the rough base is forced down upon the brain where that is in such very close contact with it. This, we can see, does not oppose the other explanation, but it is required in addition to explain the constant relation of *contre-coup* to the anterior and middle fossæ in falls on the back of the head. Mr. Hilton, however, pointed out very ingeniously that the ridges of the skull, which appear to adapt themselves to the sulci of the brain, converge and carry the force towards the clinoid processes, which are near those bruised parts of the brain. We believe this too is an important part in the sum of causation.

Sometimes the deep-seated parts of the brain suffer hæmorrhage in blows on the head. This is very important in medico-legal practice, for after an injury to the head it may be said the person died of apoplexy. To be guided rightly here we must remember that the chief seats of spontaneous hæmorrhage are about the *loci perforati*, *i. e.* the neighbourhood of the corpus striatum, and that such apoplexies rarely occur without granular disease of the kidneys, or disease of the heart, or aneurysm, or other disease of the cerebral arteries; and, again, the effusion is always large, generally two to four ounces of blood. On the other hand, the deep-seated bleedings from injury to the head are usually of small size. We have never seen one larger than a walnut, and that but once. In a case by Casper the amount was four drachms, but these effusion are generally small, and often multiple, so that several will be found scattered through the brain, if searched for carefully. They have the appearance of little beads of black blood-clot, the blood being generally held within the perivascular sheath and thus made to keep a circumscribed roundness. On account of their number these deep-seated extravasations are sometimes distinguished from the superficial as "diffused ecchymosis," the superficial being called "circumscribed ecchymosis." They do not generally occupy the usual seats of apoplexy, though exceptional cases have occurred of effusion into the corpus striatum caused by falls, but then it must be doubtful whether an apoplexy did not precede and cause the fall, as in any such case we should be inclined to believe. By careful attention to the circumstances of each case we shall usually be able to come to a sure conclusion, but this is one of the points that try the quality of a morbid anatomist.

No doubt these lacerations of the brain frequently occur in railway accidents, &c., where persons striking their heads violently suffer concussion at the time, and have cerebral symptoms for months afterwards. Judging from the recovery of such persons, and knowing what happens when the head is struck, we think a considerable amount of superficial injury may be repaired. Sometimes, on the cineritious substance on the surface we find spots of a dark yellow or brown colour, and, on minute examination, we discover in them remains of extravasations of blood, with some granule masses, showing that an irritative softening process has been going on. Probably no new cerebral substance is produced, but an indentation or cicatrix finally remains. In greater degrees of injury of the brain we believe that the ventricles are torn open much oftener than is generally taught; Mr. Prescott Hewett has collected several cases showing that the lateral ventricle may be opened into continuity with an external wound. The third ventricle is, no doubt, often torn in fractures running though the sphenoid bone; for we know how delicate is the *lamina cinerea*, which closes the third ventricle. Such laceration may lead to flow of ventricular fluid from the pharynx, or if the Eustachian tube and tympanic membrane be fissured, from the external ear, and that even without any fracture of the temporal bone. If the fracture is through the petrous bone and tympanum, the fragments drag upon the pneumogastric nerve; we have observed that this nerve has a close union with the walls of the fourth ventricle, so that a pull on the nerve always immediately tears open the ventricle. Thus, if the petrous bone be broken into the tympanum, with a slight laceration of the dura mater, and the nerve tear the ventricle, there is at once a road opened from the ventricle to the external ear, by which the ventricular fluid escapes. We must not fail to notice that sometimes the jugular fossa is so close to the tympanum that the septum is most easily burst; a common pin will go through it with scarce any effort. This relation has been scarcely sufficiently noticed, perhaps because it is not constant. Sometimes the septum is thicker; we must remember the variable size of the jugular fossa. No doubt this is the most frequent source of bleeding from the ear in fractured base.

The severest cases of injury produce *hernia* or *fungus cerebri*, as can be seen in our specimens where, after a fracture of the skull and injury or sloughing of the dura mater, an inflammatory exudation takes place, and this, mixed with the softened cerebral substance, protrudes from the surface; if cut off, it is found composed of brain and inflammatory products. Subsequently granulations may form on the surface, and a cicatrix be produced, uniting together the cerebral structure, membranes, and integument; but too often we find an abscess under the hernia, causing its protrusion, and ultimately proving fatal. Various other injuries, which may arise from fractured skull, we need not mention, as they are different in every case; but it may be as well to remember that, as we just now said, we may meet with a laceration of the septum lucidum, and extravasation of blood into the ventricles. We must especially bear in mind the occasional injury to the brain by perforation of the orbital plates, as with a tobacco pipe or umbrella stick; in such cases there may be no sign of the severity of the injury, until meningitis sets in. A fall on the nose may break the ethmoid by the impact of the nasal bone, and so cause meningitis.

**Congestion and Anæmia.**—These are conditions we have already spoken of in connection with the pia mater; for the brain shares the congestion of its membranes. We must, therefore, not attach too great importance to fulness of the vessels, which implies often merely a dependent position of the head, or that the person has died with some pulmonary obstruction, all the organs alike being full of blood.

**Hæmorrhage.**—Of hæmorrhage from injury we have just spoken, also of meningeal hæmorrhage, and hæmorrhage into the ventricles; and we must now consider hæmorrhage into the substance of the brain, which constitutes the more common form of apoplexy. First we will allude to capillary bleeding, where the blood is seen bursting forth from various parts of the substance: if a section be made in this state, not only are the *puncta vasculosa* or divided blood-vessels seen, but there are spots of ecchymosis where the blood has actually escaped into the tissue. In the same way, on the interior of the lateral and fourth ventricles these spots may be observed, and amount sometimes here to considerable extravasations into the tissue. This condition of the brain denotes a diseased state of the blood or the vessels generally, and is found in purpura, idiopathic anæmia, and sometimes in fever; also where obstruction has arisen in the sinuses. We think it interesting in connection with paralysis, which may come on in cases of fever shortly before death; we have then found this state of capillary apoplexy to exist. It is often present also after the convulsive attacks occasionally observed in typhus.

But *apoplexy*, as generally understood, signifies those cases where blood is effused in some quantity into the *substance* of the brain. We may here remark upon the ambiguity of the term apoplexy; it was originally employed to denote a disease where the patient was struck down senseless, whether this were due to an effusion of blood or other internal cause, and in this sense it is still used by some writers; but, as the cause of such sudden attack was often due to an effusion of blood, the term apoplexy came to be used by many as synonymous with effusion, so that it was carried over to express similar effusion of blood in other organs. Without commenting upon the absurdity of this, we must use the terms as they are generally employed, and as regards the brain, to avoid confusion, adopt the term *sanguineous apoplexy* for the hæmorrhage we are now speaking of. It arises from the rupture of a blood-vessel, owing to the strain on its walls becoming greater than their strength will endure. The arteries of the brain are thinner than other arteries, and the substance of the brain is so soft that it but feebly supports them when weakened. Again, we have several times spoken of the peculiar resistance to the freedom of cerebral circulation which arises through the means taken in the longitudinal sinus to prevent too easy an emptying of the brain by gravitation of its blood. Experience shows that the cerebral vessels will endure the strain upon them so long as their structure continues healthy; but more or less disease of the coats of the vessels is nearly constant after middle life, and is even frequent in early life. This disease shows itself in the cerebral arteries of all sizes, and in at least two different ways. The greater vessels about the base and the perforating arteries show chronic deforming arteritis, such as we have already described, but the smaller arteries in the cerebral substance suffer a degeneration which, though essentially similar, has a somewhat different appearance, perhaps only because as the change is microscopic, its detail becomes more conspicuous, and takes a greater share in our description. In these little vessels the nuclei of the outer coat become fatty, and this coat becomes fibrous and dilates; but the proper coats do not widen, so that they run through the dilatations and are separated from the outer coat by some interval. Dr. Handfield Jones, who has given much study to disease of the cerebral vessels, says that atheromatous patches may soften, and the fatty matter set loose block up the artery; also that clots may undergo a change and block the smaller vessels with fibroid material, granules, and hæmatoidin. For some months we examined the arteries of a portion of the *corpus striatum* in every case, and we found it was quite exceptional to discover complete freedom from this change after the thirty-fifth year of life. Sometimes the whole of the coats are dilated and fibrous, forming indeed small aneurysms, such as were described first by Messrs. Charcot and Bouchard. Dr. C. Turner found them mostly fusiform and made up of all the coats. They have lately received much attention as sources of cerebral hæmorrhage, and no doubt they do cause minute hæmorrhage by their rupture; but we think the very general existence of the earlier stages or lesser degrees of this affection in the later years of life has not yet been recognised so fully as it will be. The bleedings from such minute vessels must be small, often the rupture only affects the proper coats, so that the blood passes into the space between these and the adventitious coats, thus forming minute dissecting aneurysms; some of the "miliary" aneurysms of Charcot and Bouchard are probably of this nature. Their sections appear as little beads of black clot, perhaps partly decolorised at the surface. Similar little beads are found about great apoplexies, and in softened patches of brain, through a like rupture of the proper coats of small vessels, while the outer coat still holds its continuity; but these small aneurysms do not of themselves, we think, give rise to great apoplexies. Dr. C. Turner also came to this conclusion. He had found diseased arteries in connection with aneurysms, but the latter had not more frequently ruptured than the diseased vessels. They are important rather through the degeneration of tissue that accompanies their formation, and through the accumulation of the effects of rupture of many of them in softened brain, &c. As disease of the vessels becomes more extensive in the course of life, so necessarily with it the tendency to apoplexy increases with age, and, *cæteris paribus*, the older the person the more liable is he to an attack; but the great predisposing cause is granular disease of the kidney. This is, indeed, so frequent an associate of apoplexy that no surprise will be felt at its almost constant repetition in the cases that occur in the post-mortem room. Thus, of the last seventeen cases of fatal apoplexy we inspected, fourteen had granular kidneys.

The hypertrophy of the heart in this disease we have already spoken of. It must be remembered that this hypertrophy is associated with disease of the arteries, and is not combined with dilatation as in primary heart disease; so that it actually augments the pressure in the vessels. Hence it is that the cardiac hypertrophy of granular kidney tends to rupture the arteries, whilst the arteries are also bad; and we thus have two conditions in granular kidney, either of which tends to produce apoplexy. The strong heart breaks the weakened arteries.

The special seat of apoplexy is where large arteries enter the brain to supply those great masses of ganglionic matter—the corpus striatum and thalamus—which subserve the functions of the extremities. It is to be noted that rather large vessels pass from the carotid and middle cerebral arteries directly into the brain at the *locus perforatus anticus*, a spot just under the ganglionic masses we have named, whereas in nearly all other parts of the brain the arteries break into a wonderful network in the pia mater from which only very minute arterioles pass into the cerebral substance. A very large proportion of all apoplexies, more than three fourths, occur about the perforated spot, in the corpus striatum, or thalamus, or both, or just outside or between them. The blood passes up the external capsule outside the lenticular ganglion; ploughs up this district, pressing on the corpus striatum and thalamus; or tears open the outer part of the former ganglion, making its way to the ventricle, or more rarely breaks its way through to the convolutions on the surface. Another rather frequent position relatively to its size is the pons, in which we met with five fatal apoplexies in three years, as well as some which had been recovered from. The rest of the brain is in all parts about equally rarely affected, except that apoplexies in the medulla oblongata, and in the corpus callosum, are exceedingly rare, while in the grey matter of the surface they are more common.

Indeed, it may be shortly said that apoplexy affects the vessels of grey brain matter rather than white, and deep-seated grey matter rather than superficial. An ordinary fatal apoplexy displays a degree of forcible destruction which proves the bleeding to be from a vessel of considerable size; no small vessel would carry the force that is exerted, when, as we often see, two to four ounces of condensed clot are found effused (three or three and a half ounces is a common amount in a fatal apoplexy in the corpus striatum). The brain tissue is then torn in a most terrible way, fragments of it are mingled in the clot mottling its outer part, strands of the fibres are rent across, and their torn ends, half an inch to an inch across, are often plainly seen projecting. In the tissue around the chief effusion many small vessels are torn, and generally in such a way that the bleeding from them is confined by their perivascular sheaths, producing on section those small bead-like knots of blood we have before mentioned. The characters of such a violent laceration across the fibres infer a bleeding from a considerable vessel in these cases, and we do not think miliary aneurysms would produce such an effect. We sometimes see smaller apoplexies, and these usually run in the course of the bundles of nerve-fibre, being restrained by their resistance. This is generally well seen in apoplexies in the pons which evidently separate the fibres in most instances. It is interesting to notice that the parts which chiefly suffer apoplexy are those that chiefly undergo softening. For this, like apoplexy, is not a primary disease of the nervous elements, but an accident to it through imperfections of its blood supply; thus apoplexy may occur on one side, and softening on the other. It is when the grey matter actively serves the limbs and demands a rich supply that the tribute for all this extra wear and tear has to be paid, the limbs themselves suffering palsy through the stress caused on the vessels by their own excessive demand.

Apoplexies are usually single and are thus distinguished from deep-seated effusions due to injury, but we have thrice seen apoplexies in the pons coexist with great apoplexies in the corpus striatum; on one occasion the apoplexy in the pons was double. In these cases the effusions were evidently simultaneous. Dr. Hughlings Jackson has collected several examples of simultaneously multiple apoplexy, in one of which there were four effusions in distinct parts of the brain. These cases are, however, quite exceptional, and all the effusions are in the ordinary seats of apoplexy; we do not think that there would arise serious difficulty in distinguishing between such cases and diffused contusion of the brain, in which usually there is some obvious superficial bruising, and the effusions are not in the common seats of apoplexy; sometimes, however, the difficulty might throw us back on the history and associated conditions, as in Casper's case, in which, with a considerable effusion of blood in the pons, there was a free arachnoid hæmorrhage, like an apoplexy. In very great apoplexies the effusion is apt to reach the inner surface of the brain at the ventricles, or its outer surface in the arachnoid space.

When the effusion is great death occurs in the course of a few hours or a few days; the shortest time in which we have known an apoplexy to be fatal through compression without shock was forty minutes; in this case the floor of the fourth ventricle was torn up to pulp, and the ventricle itself was full of blood; another case, limited to the pons, lasted two hours. In very rare cases death occurs instantaneously, as in an instance recorded by Dr. Dickinson; we quite agree with him in referring the suddenness of the death in such cases to an abnormally precarious state of the heart through fattiness, &c., of that organ.

Although the nerve lacerations do not cause rapid death, yet various kinds of injury will do so; this is probably due to shock or concussion. For example, a pistol-shot wound is often immediately fatal, and we know of the case where a man was struck by another with his fist behind the ear and was killed on the spot. There was no mark of external injury, but blood was effused at the base of the brain.

When the effusion is less in amount or less critically situated, changes occur of a healing nature. The serum must, no doubt, be very quickly absorbed, if, indeed, it ever appears in quantity; we rather believe that the serum is removed during the progress of the effusion, for we have never seen any serum but always remarkably firm clot. The clot changes as well as the wounded tissue. The clot becomes decolorised, turning pale brown or buff from the surface inwards. Meantime it dwindles gradually away; as it disappears the space it occupied is now seen to be crossed by threads, the remains of vessels, &c., that escaped laceration. After many months the clot is all gone, and nothing is left of it but some hæmatoidin crystals and grains which remain clinging in the cicatrix, but even these may at last quite disappear. All this while the injured texture is forming a fibrous scar tissue close around the clot, and this may develop well, and produce a cyst-like fibrous sac, the "apoplectic cyst;" when the clot is gone, this cyst remains filled with clear fluid, crossed, as we stated, by threads. We have several specimens of such cysts at one, two, three, four, and eight years after the apoplexy. The wall of the cyst is made of wavy connective tissue including blood-vessels; it is believed to arise from the neuroglia by proliferation.

Sometimes matters take a less favorable course, and the tissue around the clot undergoes subinflammatory softening, perhaps with fresh effusions of blood; the texture then, for a distance around, is found permeated by those "granule globules" of which we shall speak under "Softening." It is said by some, as Niemeyer, that abscess may form at the seat of an apoplexy; we know no facts to support this statement. It certainly is remarkable that severe spontaneous injury to the brain should so very rarely indeed excite the higher inflammations, when traumatic hæmorrhage so often causes suppuration.

The end of the most favorable cases is, then, that the clot is all absorbed, and the cyst-like sac which surrounded it is found with a little fluid in it, or almost or quite closed, but not with its walls adherent. There is danger of recurrence of the accident, and it is far from uncommon to find three or four apoplectic patches in various stages.

We must now notice that some of the worst apoplexies are due to relatively large aneurysms of the greater cerebral arteries, such aneursyms reaching the size of a pea or horse-bean, or even becoming as large as a cherry. It is interesting to notice that they are apt to occur in young persons; about half have occurred before the age of twenty. It is highly probable that in young persons, where there is no general disease of the vessels, if the apoplectic portion of brain were thoroughly examined, an aneurysmal condition might often be found, for such has sometimes been discovered when not expected. These are usually associated with emboli and endocarditis. Gull and Brinton collected a large number of cases of aneurysm of the cerebral arteries. They found aneurysm of the basilar the most common, second that of the middle cerebral, and then the anterior cerebral and posterior communicating. Dr. Bristowe has given a remarkable case of a large aneurysm of this kind within the cerebellum. We have seen carotid aneurysm in the cavernous sinus burst into the lateral ventricle.

Besides these greater apoplexies, we often meet in old people with bad vessels, and especially if they have Bright's disease, with examples of numerous, small, brownish, soft spots, showing the remains of old effusions, &c. Such patches are the causes of slight numbnesses and weaknesses of the limbs in these people. They are often associated with fibrinous plugging of the vessels whether from emboli or thrombosis.

The so-called *serous apoplexy*, or water-stroke, is a disease of which we know little; it was a term formerly used to designate those cases of very speedy death with coma where no blood was discovered in the brain. It was the occurrence of these cases that induced Abercrombie to suppose that the fatal pressure in apoplexy is always within the vessels, and that the escape of blood into the brain is only an accident of some cases. If we read the cases given by Abercrombie of the state of health of his patients, and all the accompanying symptoms, it can leave no doubt on our minds that many of them were instances of Bright's disease. Apart from such, however, we do occasionally meet with cases of very rapid death from evident cerebral affection, and yet scarcely anything is found to account for it on examination; but in these there is no increased effusion, so that the term serous apoplexy cannot be used; it is a term we never adopt, and scarcely do we know to what class of cases it is applicable. Some describe, with Niemeyer, an acute œdema of the brain in Bright's disease, that is, an effusion of serum into its mass from unknown causes, but we have not found anatomical evidence of this. The death in these cases is probably chemical, and not mechanical. It is possible that some instances may have been of a kind such as we have mentioned under chronic hydrocephalus; where, for a long time, a chronic inflammatory action has been going on in the ventricles, and yet death occurred suddenly, and very little was found except the increased fluid in these cavities, which had no doubt existed for a long time.

*Cerebritis and softening.*—As one of the results of inflammation of the substance of the brain is softening, it is often difficult to determine which of these expressions is the more appropriate to use in any individual case. As all instances of softening are not due to an inflammatory process the French use the term *ramollissement* in a very general sense whatever the determining cause may be. In some cases it is evidently due to inflammation, whereas in others, when the brain decays from disease of a blood-vessel, the softening is merely one of disintegration. It should be remembered that softening in the popular sense merely signifies a permanent impairment of the functions of the brain when the mind is weakened, so that when the medical man uses the term to his patient's friends he by no means implies softening in its strict pathological sense. In inflammatory softening a considerable amount of hyperæmia may be present as well as effusion of blood products; consequently the affected part is of a pinkish colour, and the term *red softening* is given to it, whereas when softening has occurred from failure of nutrition the part has remained white, and the term *white softening* is used. The term *yellow softening* is also employed, but this condition is only a later stage of red softening, when the hæmatin has undergone chemical changes.

If we make a post-mortem examination and find softening of the brain, how does this condition display itself? When a section is made through the brain we see the hemisphere, or a part of it, presenting a peculiar appearance, looking and feeling pulpy like a piece of blancmange; the surface is not smooth but rough, and on section presents a broken surface; the cerebral substance sticks to the blade of the knife, and if we stir it about we can form it into a paste. If the softening occupies only a few spots, these are indicated by an equivalent number of broken surfaces; and if it has proceeded a stage further, the brain matter is found quite broken up or may be semi-fluid, so that a stream of water allowed to flow upon it leaves a depression or hole in the substance. Sometimes, when this absorption has taken place during life, a hollow space is found, containing only débris of blood-vessels, and a fluid like lime-water. Such extreme cases, however, are always chronic and the result of decay from disease of the blood-vessels. There can be no doubt that in a very large number of cases softening is due to deficient blood supply, either arising from a general degeneration of vessels, or from a local thrombosis. In old persons distinct spots of softening of the brain clearly have their origin in these vascular changes; but it is remarkable that in younger persons, where spots of softening may be found in various parts of the brain, the vessels are also diseased, thickened, and atheromatous. The arterial disease here, however, is confined to the brain, and is not shared in by the vessels throughout the body as it often is in Bright's disease. Such cases mark the pathology of the softening as strictly local, and associated with vascular changes in the brain only. A similar fact may be observed also in the purely local degenerations found in the vessels in various liver and kidney diseases. It is difficult under these circumstances to pronounce the vascular change as the primary one. Nevertheless, thrombosis and embolism are amongst the commonest causes of softening of the brain. The thrombosis arises usually from a previous disease of the vessel, either atheromatous or syphilitic; but it may occur from some unknown change in the blood itself. The embolism may be a simple fibrinous clot or may be infective, and so leading either to softening or abscess. In the first case the brain suddenly becomes anæmic, is then tinged with blood from venous congestion, and subsequently breaks down. In the red or inflammatory softening, the disintegration is not so great; here the affected part may be plumper, and of a pinkish colour. A later stage would be the yellow softening, which is apparently due to an alteration in the colouring matter of the blood. Although we make these distinctions between the red and white softening, as indicative of inflammation or the contrariwise, we cannot say that they are absolutely characteristic, for a difficulty arises in such a case as tubercular meningitis, where a very marked softening takes place in the central parts of the brain, the septum lucidum, fornix, and adjacent parts being so broken down that they are almost diffluent, and yet they are perfectly white. The microscope shows merely disintegrated nerve-fibres and granular corpuscles, but considering that the disease of the brain has been inflammatory, it is not too much to assume that the changes in the ventricles are of a similar nature.

It is convenient to divide cerebritis or encephalitis into *general* and *local*, and these again into the *acute* and *chronic* forms.

*Acute general cerebritis.*—It is very possible that this may be more common than is generally supposed. We find, both in traumatic and idiopathic inflammations of the brain, the products of the process both on the surface and within the ventricle; and therefore it is not unlikely that some change, be it only a molecular one, has taken place throughout the whole substance of the organ. We occasionally meet in such cases with fresh-formed cells and points of hæmorrhage indicating that in all probability a very general inflammatory process has occurred. Moreover, although little trace of such inflammation is found after death in such fatal affections as acute mania, or in the hyperpyrexia of cerebral rheumatism, we cannot say that no inflammation has occurred. For if such cases were not immediately fatal, no doubt in a very short time the disintegrating process would be evident. There are now and then indications of this. As an example of general cerebritis we may mention the case of a young man who was admitted into the hospital, supposed to be suffering from fever on account of the torpid state into which he had fallen; he never spoke, but lay perfectly motionless in bed with his eyes open, apparently watching those around him. His illness lasted only a few weeks, and after death the whole of his brain was found soft and pulpy, whilst some parts were semi-fluid.

Although it is necessary and convenient to separate, whenever we are able, the different structures which are inflamed, yet it sometimes happens that we find the whole of the cranial contents, including the cranium itself, the subject of an inflammation; the internal surface of the calvaria being covered with granulations, the membranes thickened, and the whole brain showing spots of softening or cysts.

*Acute local cerebritis.*—This we have already spoken of under the name *red softening.* It may arise from injury, from embolism, or from incipient pyæmic inflammation. When an embolus is thrown into a cerebral artery from an ulcerated endocardium, the part of brain fed by that artery may be found in any stage of inflammatory change, and be red, soft, and swollen; or after a time be hardened by the intermixture of nucleated fibres, the originally red softened tissue also becoming brown or ochreous yellow from a change in the pigment of the blood. A local inflammatory softening is also found, not only in connection with obstructed vessels, but with apoplectic effusions, this being often surrounded by a circumscribed cerebritis. The middle cerebral is the vessel most likely to be affected by embolus, and the left slightly more frequently than the right. Next in frequency is the posterior cerebral. The anterior cerebral and cerebellar arteries are more rarely blocked.

A very remarkable case occurred in the hospital, where the grey substance was the part most affected by the disintegrating process. A woman, after striking her head, suffered from cerebral symptoms of an obscure character, and after death there was found disease of the whole surface of the right hemisphere; this was of a yellowish colour, soft, and had the appearance of being worm-eaten, or in the state the French call *état criblée.*

*Chronic general cerebritis.*—We use this term where a large portion of the brain is affected, and thus distinguishable from the cases where the inflammatory products are present in small patches. In most cases of this kind the membranes have been involved, and therefore the expression meningo-cerebritis would be the more appropriate term. A young girl was sent into the hospital for hysteria, owing to the indefinite nature of the nervous symptoms under which she suffered. She was perfectly helpless, could scarcely move her arms, and spoke and swallowed with difficulty, having also a stupid expression. She made no complaints, and generally laughed when spoken to. After death the pia mater was found thickened, of a yellowish colour, and adherent to the brain; this was more especially the case over the frontal lobes. A section of the brain showed it to be hard throughout, but this was more especially the case with the anterior lobes, which could be grasped in the hand like a hard tumour. The colour and appearance was otherwise peculiar, both grey and white substance being changed into a brick-red colour; this was more especially the case in the front part, where the distinction between the cineritious and medullary portions could not be recognised; at other parts the grey matter was twice its natural thickness. The red tint extended inwards to the roof of the ventricle. The microscope showed complete disintegration of cerebral structure, the nerve-fibres had almost disappeared, and their place occupied by granule masses, vessels, and fibrillated tissue. There was also lately in the hospital a girl of fourteen, who was sent to the hospital in a perfectly helpless condition, both in body and mind, so that those who first saw her thought her to be an idiot. A year before her death, however, she was an intelligent child. She lay quiet in bed in a state of vacuity, until she died. It was then proved that she had been the subject of a general meningo-cerebritis. The surface of the brain had lost its transparency, the arachnoid was thick and opaque, and the pia mater was firmly adherent to the cineritious surface, so that it was impossible to raise it without tearing the brain-substance. The brain as a whole was much firmer than natural, it felt hard to the touch, and the septum lucidum could be stretched out firmly without laceration. There were no tubercles in any part, nor could any cause be found for the occurrence of this general chronic inflammation of the membranes and substance of the brain. In these cases of chronic inflammation it will be observed that portions of the brain are harder than natural; this is due to a production of connective tissue having its origin in the neuroglia. Dr. Bright, in his medical reports, relates the case of a little girl who for a year before her death lay in a perfectly motionless and senseless state, with her limbs stiffly extended and without the possibility of making the slightest movement. After death the white substance of the brain was found almost as hard as cartilage, and a stream of water washed off the grey matter, leaving the convolutions modelled on the white substance beneath, giving it the appearance of a wax model of the brain. The ventricles also looked as if they had been modelled in wax.

*Chronic disseminated cerebritis—Sclérose en plaques—Insular sclerosis.*—This is found throughout the brain and spinal cord, or more rarely limited to either. It appears in the form of circumscribed patches, generally circular on section when small, but more irregular when large, and then reaching the size of a hazel-nut. They have much the appearance of grey cerebral matter, but are tough and firm, the whole brain also being usually much firmer than natural. When small they appear to surround a congested vessel, but as they grow larger this is not evident. They arise in points and spread; at first being of a grey colour, but becoming more curdy in appearance. They rarely invade the cineritious matter. The microscope shows that in these patches the nervous elements are greatly wasted, and that a fibrillar hyaline material is present, in which may be found also granule masses and amyloid corpuscles. The axis-cylinders remain for a long time intact. Such patches may be found not only in the brain, but extending through the crura, pons, medulla, and cord. We have seen the pons almost entirely sclerosed into a grey mass and the aqueduct of Fallopius surrounded in a very curious manner. When the disease spreads it is called diffuse.

*Abscess.*—Another result of cerebral inflammation is suppuration; this, however, cannot be looked upon as the ordinary extreme of inflammations from common causes. These, as we have just shown, result rather in indurations from exudations of lymph, or softenings, or cyst formation. Abscesses of the brain arise either through suppuration of the bone due to injury or disease, or else they are part of a general pyæmia; as to this we must observe that they may be secondary to suppurations in other parts where the general pyæmia is otherwise slight and transient. The cerebral abscess may be the only secondary suppuration present after an operation. Our own opinion agrees with that long ago advanced by Sir W. Gull, who concluded that abscess of the brain does not occur spontaneously. We have twice seen examples of the occurrence of abscess of the brain from injury without fracture, as mentioned by Mr. Prescott Hewett; in these cases if the abscesses should become encysted, as they sometimes do—we have one which came from a case of fracture of the skull eleven weeks after the injury, when the fracture had healed—the blow on the head might easily be unreported and so the abscess appear to be spontaneous. In any case of abscess of the brain, suspected during life or found after death, we should seek carefully to find some primary seat of suppuration. In a late case of cerebral abscess we were nearly in despair of explaining its origin, having searched in vain all ordinary situations of suppuration, but we found that the right bronchus, from near the end of the trachea to the second bifurcation, had its mucous membrane ulcerated away, so that the cartilages were in parts exposed and necrosed. Now, the cerebral symptoms had masked the pulmonary in this case, and it would have been easy to have passed by what was certainly the prime cause of the abscess. It is remarkable that cerebral abscess has often been met with as secondary to bronchial cavities and bronchial gland suppuration. We have known abscess of the brain arise from ligature of a pile, and from excision of the eye-ball. We have never ourselves met with an example of non-traumatic cerebral abscess where a careful search has not at last revealed the presence of primary suppuration in some other part of the body, and we should consider such a case to be an unsuccessful examination. The first stage of the abscess is sometimes met with as a deep-coloured red patch of softening, and is an evidence of its association with general pyæmia. After the development of pus the abscesses will differ according as we find them recent or old; if recent the abscess wall is irregular, or even ragged looking, and has no proper membrane, its contents are more liquid and of a pale-greenish sulphur colour, and may be fetid and black from sulphuretted hydrogen; this depends chiefly on whether the bone disease it arose from is necrotic; an older abscess has a round shape and smooth wall, which is defined by a "cyst" of adventitious connective substance. This cyst may show microscopically good wavy fibre in its middle or best developed part, while its outer part shades off into the brain tissue around, and its inner surface has the character of pyogenic membrane. A very old abscess, which had existed eighteen months, had its cyst so thick and firm that we could lift it out of the brain tissue around. The man had had pyæmia from lithotomy in this hospital eighteen months before; he recovered and had worked for sixteen months in the fields, when he came in again and died of coma after a convulsion. The contents of these old abscesses are green, alkaline, and even ropy; the microscope shows the pus-cells to have degenerated almost entirely away in old specimens; they may last very long. In one case we knew there was every reason to believe that such an encysted abscess had been present in the brain for many years. This prolonged innocence and the gradual degeneration of the abscesses make it probable that some of the old calcareous relics found occasionally in the brain may have been due to former abscess. These old abscesses were single in each case, but sometimes, especially in acute pyæmic cases, there are several or even numerous abscesses. Multiple abscesses are generally speedily fatal, but we had one remarkable case in which there were more than twenty encysted abscesses in the brain of a lad who had worked as a shorthand writer to within five days of his death. Abscesses are by far more often found in the white matter than the grey matter of the brain. Gull and Sutton say that of seventy-four cases collected by them only four occupied the corpus striatum or thalamus. Abscesses belong mostly, indeed, to those great masses of white matter which constitute the *centra ovalia*, whose function is shown in softening and tumour to be less distinctive, hence abscess is so frequently latent; a man with ounces of pus in his brain may continue to do good mental work and have full use of his limbs.

A great number of abscesses are produced by old caries of the ear: what it is that superinduces the abscess after so many years is often not at all clear, but there may be obvious recent extension of the disease producing necrosis of the petrous or mastoid bone, and so involving the dura mater and sinuses, although sometimes the dura mater is not involved. The abscess usually lies close to the seat of its cause, whether this be injury or caries, but a layer of brain tissue often, not always, intervenes. This neighbourhood is important to remember; it has even allowed the successful evacuation of pus by operation in cases of fracture, and sometimes the abscess near the ear has discharged through the temporal bone (otorrhœa cerebralis). We have mentioned the occurrence of abscess under, and causing, *hernia cerebri.*

Lastly, the tissue around an abscess of the brain for a greater or less distance is generally softened by extension from the focus of inflammation.

**Syphilitic Disease of the Brain.**—We have given some cases of general meningitis which occurred in syphilitic subjects. These are rare, and indeed some doubt will perhaps attach to them, until the observations are more extended. We will now speak of characteristic syphilitic gummatous disease of the brain. Quite contrary to the meningitic cases we were alluding to, the peculiarity of this form of disease is its circumscribed extent. It always appears as a tumour-like nodule which is generally single, though occasionally there are several. It by far most commonly affects the surface of the brain, so that it is quite rare to find syphilitic disease of deep parts unconnected with the surface. Virchow makes the interesting remark that the parts affected are those most prone to suffer in mechanical injuries to the head, as though the effects of such injuries predispose the part to suffer. The syphilitic gumma has generally an irregular figure, representing indeed usually the form of the part attacked and changed by it. The consistence is firm, the colour pinkish red, like pale flesh at the outside, with a caseous centre which represents the older decaying part of the growth; or this caseous part may be in the form of a mottling of the central area through coalescence of its originally distinct patches. But always the caseous portion forms the greater part of the patch; the tissue around is commonly in a state of yellow softening, but the patch has no defined surface towards this.

It usually, by being formed in the pia mater, causes adhesion between the membranes, and thus the dura mater is found fixed to the brain. On the inside it involves the grey substance. A very favourite seat is about the Sylvian fissure, and here it invades or obstructs the middle cerebral artery. This accounts for the paralysis which accompanies the convulsive fits set up by cerebral syphilis.

Gummatous masses may also be found all over the base, posterior lobes, and cerebellum, causing adhesions to the dura mater with thickening of the nerves and vessels. In these cases there is no characteristic change in the vessel; all the coats may be involved, and the vessels matted together externally, and the interior occupied by thrombosis. The brain is not often affected in congenital syphilis, but Dr. Barlow had the case of a child ten months old where there were thickened patches of pia-arachnoid with adhesions to the brain.

It is far from easy to distinguish these patches from tubercle on the one hand, and from sarcomatous tumour on the other. But a cerebral tubercle is generally deep seated, and has a spherical form; it is more distinct from the parts around than the syphilitic growth. It is more entirely caseous, and its cheesy part has a greenish hue, and a more curdy aspect; the margin of vascular tissue is very narrow. In each of these points we see that there is a distinct contrast with the syphilitic gumma.

From tumours syphilitic gummata are known by the larger proportion of caseous change they have undergone, as well as by their less defined surface, and by the less perfect development of their elements. In syphilitic formations the cells are always ill-bred looking, short and small; the spindle- and round-cells have a granular unclearness and imperfect nuclei; while in sarcoma and myxoma of the brain it is remarkable how beautifully developed are the individual cells in the undecayed part of the tumour.

In all the cases hitherto described as occurring within the brain there has been traced a connection with the pia mater on the suface.

**Morbid Growths.**—The list of cerebral tumours is a list of tumours generally; for every named kind of tumour has been found in the brain. Our notice of each must then be short and limited to those points special to the brain. They probably may have their origin in the membranes, vessels, or neuroglia.

*Carcinoma.*—This we have found in a most typical form in the brain, secondary to scirrhus of the breast, &c.; its cells are apt to form glomerules in a more hyaline and less fibrous meshwork than that found in the breast and other parts, but carcinoma appears to occur also as a primary affection.

*Myeloid* tumours also have been found in the brain; several such cases are in the Pathological Society's 'Transactions.'

*Sarcoma.*—We meet with very beautiful spindle-cell tumours in the brain, the spindles peculiarly long and well formed, and easily extricated from their neighbours; such tumours are generally encapsulated; the capsule closely resembles pia mater, and often is connected with this membrane. Hæmorrhage into these tumours is frequent.

*Myxoma.*—This is occasionally found. According to Virchow heteroplastic myxomata form very often in the nervous tissue; he associates this with the close alliance in nature between the soft neuroglia and mucous tissue. They form large tumours, of a soft translucent gelatinous substance, and by the resolution of their central part they may break down into cyst-like cavities full of slimy fluid. He gives instances in which they appeared to be congenital.

*Melanosis* of the brain has occurred occasionally as part of a general melanotic disease; it is very striking because of its black colour against the white of the brain. But, as we have said before, melanotic cancer, with melanotic sarcoma, is only ordinary cancer pigmented.

*Glioma.*—This is the proper tumour of brain tissue, and developed from neuroglia and cells. It is distinguished from the others by having no capsule, but merging indefinitely into the tissue around. It is firm and tough, otherwise very like brain tissue, but more pellucid. It is very liable to undergo successive hæmorrhages, so that blood in various states is present in the tumour, giving brownish or purplish colours to the central parts of it, or ending in false cysts there. The microscopic structure is very variable. Sometimes there is scarcely a trace of structure except fine delicate filaments forming a feltwork, then we get cells small or large, scattered in such a tissue, resembling sarcoma very closely (and being indeed the sarcoma of brain). We have twice met with a very peculiar form of it, in which the cells were like those star fishes that are called "heads of Medusa," each having a beautiful conspicuous cellular body, with countless bushy stellar rays running out of it, very like those of a bone-corpuscle. The tumours in these cases were scarcely discoverable by their colour, only by their consistence and the pinkish clear swelling of the affected part. It is to these tumorous swellings or "infiltrations" of parts that the question must always apply whether the tumour is not due to a chronic inflammatory irritation, whether, in short, it is not a chronic inflammatory thickening of the brain-substance. The growth often has no defined limits, so that, when the pons is affected, it appears simply overgrown.

*Osteoma.*—This has occasionally occurred in the brain. Such bony tumours are of slow growth and may be almost latent. Thus, Ebstein fully described an osteoma of the cerebellum which had produced no symptoms.

*Heterotopia cerebri.*—We have only once met with a decided example of this curious condition, a development of true ganglionic grey matter under the ependyma of the lateral ventricle (posterior cornu), of the size of a hazel-nut. Attention was directed to these first by Rokitansky and Virchow; they describe multiple patches in the same situation.

*Erectile tumours.*—We have ourselves never met with these. Cases are recorded; we have mentioned Mr. Morris's case of angioma of the pia mater invading the brain. Dr. Bevan Lewis met with a similar case undergoing calcification.

*Cystic tumours.*—These are very rare. The recorded cases are few; but we have a remarkable cystic tumour of the cerebellum. The cyst is as large as a small apple; the little mass on one side of it is well-formed carcinoma, and the tumour was primary. We have already spoken of cystic degeneration. We have before alluded to the posterior cornu of the lateral ventricle cut off and forming a cyst. Dermoid cysts have also occurred in the brain. They have usually been connected with the dura mater and have no doubt sprung from some remnant of embryonic tisssue. They contain sebaceous masses and hair, and sometimes have caused death by suppuration.

*Cholesteatoma.*—A tumour composed of cells and cholesterine is sometimes found at the base of the brain. The cell structure alone would constitute it an *endothelioma.*

*Psammoma* we have already mentioned under membranes.

*Calcareous masses* are met with sometimes in the brain, and are probably old decayed tubercles or dried-up abscesses.

In considering generally the question of the malignancy of a cerebral tumour, we must remember that we know the malignancy of a tumour most certainly by its infecting other parts, as the lymph-glands or viscera. Now the brain is so vital an organ that death may be thought likely to supervene before time for secondary growths has elapsed. Again, doubt and obscurity still hang about the distribution of the lymphatics within the skull; it is even questionable whether there are any lymphatics there at all. We must say, in spite of the description of lymphatics within the skull, that we have never found the lymphatic glands at the base of the skull enlarged in cases of malignant tumour of the brain, unless such tumour implicated the dura mater or bone. Thus it appears that there is not the usual channel for invasion of the glands, and hence the ordinary generalisation of cancer is thought not to occur in the brain.

*Tubercle.*—We have already spoken of tubercle affecting the membranes, and of its being especially found in those portions of the pia mater which dip down between the convolutions. When tubercle occupies the brain tissue it rarely appears in this form, but in large yellow masses, resembling milk of sulphur in colour; these are always very firm, and little disposed to break down. Occasionally we have seen one softened in the centre, but considerable softening of them is rare. These masses, from growing in a soft organ, are generally quite round; and thus a globular tumour the size of a billiard ball, or even larger, may sometimes be found. Then their surface we see by careful scrutiny to be made up of agglomeration of minute miliary tubercles which spread into the tissue around, while the advancing caseous mass involves them in its spread. Rindfleisch speaks of two forms of these tubercles, one of which is thus made up of miliary aggregations, while the other is only an almost amorphous scrofulous deposit; we believe this difference is one of age and activity, and that the latter form is an older tubercle, not spreading at the time of death. We mentioned that these tubercles sometimes set up tubercular meningitis. We believe the larger tumour of "tubercle" in the brain is the primary tuberculosis or "scrofula" of that organ, while the miliary form is always secondary, or, at any rate, is part of an acute tuberculosis.

Miliary tubercles in the substance of the brain must be very rare, for we have only once met with a few of them. Dr. Gee, however, describes a case where part of the cerebrum was softened and stuffed with minute tubercles in tuberculous meningitis.

These large *tuberculous tumours* are more frequent at the base of the brain than elsewhere, and thus are very commonly met with in the cerebellum, which is their favourite seat. Sometimes, on removing the brain, a lobe of the cerebellum may appear slightly altered in shape or size, and then, on cutting it through, a large uniformly smooth yellowish mass is seen occupying this part, but having a layer of cineritious structure over it. Tumours in this neighbourhood press upon the surrounding parts, and produce various symptoms ; amongst others they cause ventricular effusion, and give rise to conditions resembling those of chronic hydrocephalus. We can call to mind two cases of chronic hydrocephalus where these large scrofulous masses existed at the base of the brain. They are often yellow in the middle with a gelatinous material around and generally grow inwards from the pia mater.

It is truly astonishing how great a destruction of the vital parts of the brain may be insidiously affected by these chronic large tubercles. On our shelves is a *pons Varolii* with a tubercle occupying its central part to such an extent that the remaining tissue forms only a delicate, scarcely continuous film around it. Sometimes they reach an enormous size, as in another preparation, like a large potato, which was presented to the Museum by Dr. Hughlings Jackson : it is from a cerebral hemisphere. Sometimes there is more than one tubercle present, but generally one only.

The microscopic structure of these tubercles is seen at their peripheral part ; we here find often a concentric arrangement of lymphoid cells around vessels, the whole representing a considerable degree of structure. But within a few lines of the surface inwards the elements are faded away to relics. Outside is a red zone of soft tissue, often very delicate, so that the tubercle easily falls out. For an inch or so all around the brain tissue is in a state of yellow softening. Characteristic bacilli are found in them.

*Melanœmia.*—The malarial or pigmented brain is well shown in a drawing in ‘Bright’s Reports.’ The cortex has a black-lead hue and the cut vessels appear as black points. We shall allude to it again under “Spleen.”

**Hydatids.**—We have mentioned these as occurring on the surface of the brain in connection with the arachnoid, and also in the ventricles. These have always been, we believe, *cysticerci ;* but the *echinococcus* is occasionally met with in the substance of the brain, of which we have examples. We recently had a specimen of a very large echinococcus in a young girl from the Clinical ward. This reminds us that it is said the echinococcus occurs more often in early life, and the cysticercus in older people. When one is present in the brain, another echinococcus is usually present in the liver; it was so in our recent specimen, but it is not always so. Generally they are acephalous; but scolex heads are sometimes present.

### PITUITARY GLAND, OR BODY

This may undergo morbid changes, and be subject to diseases, as other parts, and thus more than once have we seen it occupied by cancer. Owing to the large cells which the glandular part of this body contains, with their numerous nuclei and pigment, it was suggested by Dr. Gull whether this body had any relation with the suprarenal capsules, which contain somewhat similar cells; we have since looked in all cases of morbus Addisonii for any disease of this body, but have not yet found any appreciable change.

Hale White met with a tumour of this body which he called *myoneuroma.* It was the size of a nut, and composed of striped muscular fibres mixed with nerve-fibres. He thought it was congenital, for tumours have been found in this body containing hair, cartilage, and muscle, which probably arose from some embryonic structure.

*Cancer* of the pituitary body has been found, as mentioned above.

*Tumours* have also been found in this body in three curious cases of the deformity called *Acromegaly* (see Myxœdema).

The PINEAL GLAND often contains calcareous sand. This, called brain sand, *acervulus cerebri,* is generally seen in grown persons along the anterior border of the gland and its peduncle (*psammoma*). It is made up of endothelial cells derived from the pia mater, choroid plexus, or other part. Endothelial globules become calcified and form the concretions or brown sand, of carbonate of lime, already alluded to.

A spindle-celled *sarcoma* has also been described. We have several times found cysts in this gland, once as large as a cherry, and once with a hæmorrhage into the interior. No symptoms were evidently referable to these changes in the gland. Two of these cysts occurred in the subjects of Bright's disease.

## SPINAL CORD

We shall consider the diseases of the membranes and medulla in succession, in the same way as we have those of the brain, though many of the remarks we have made upon the latter will equally apply here. Thus, in idiopathic inflammation of the membranes, the exudation is from the pia mater, and consequently beneath the visceral arachnoid, as in the brain; only in very acute cases of idiopathic inflammation may a little lymph be found on the free surface of the arachnoid.

But we must observe that the dura-arachnoid of the spinal canal is not quite the same in its relations as the dura mater of the cranium, for it does not serve for periosteum. It is, indeed, a special membrane of the cord. We must also notice that the arachnoid is much more distinct from the pia mater, and the subarachnoid space much more defined and regular in the spine than in the cranium. We shall again consider these parts as dura-arachnoid with arachnoid cavity, and pia-arachnoid with subarachnoid space.

As we recommended in the brain, so it is important also in the spinal cord to bring our knowledge to bear on its appearance, when the examination is first made—on section, to remember the anterior lateral tract with the crossing of the pyramidal fibres, with the exception of a few direct ones which run down at the sides of the anterior fissure, and which are called Turck's columns. Then to notice the outer edge of the anterior lateral columns called the cerebellar tract, and which appears to be associated with Clarke's columns. Then the posterior columns, divided into the funiculus gracilis, or columns of Goll, running on each side of the posterior fissure to end in the medulla oblongata, and the funiculus cuneatus, or columns of Burdach, called also the posterior root columns. Then to observe the motor tract passing downwards to the medulla from the cortex above through the internal capsule and the under part of the crus and pons. It is important also to notice the grey matter with its anterior and posterior horns and the intermediate tract on its outer side. Then on microscopic examination there should be noticed the character of the grey cells, whether dark, shrunken, or ill-formed; also whether sclerotic patches are seen, these being composed of fibrillated tissue with wasted nerve-tubules, or more recent inflammatory spots showing the degenerative process, the medullary sheath breaking up into fat-globules, whilst the axis-cylinder still for a time remains. We may remember also that Ranvier describes the nerve-fibre as constricted at intervals, each internodular portion possessing its nucleus. The myeline is thus made up of segments, although the membrane of Schwann is continuous over all. This, however, does not occur in the brain and cord, where there is no membrane of Schwann, and it must be remembered that there is no medullary matter around the axis-cylinder of the nerves of Remak in the sympathetic system. The axis-cylinders appear to be the direct continuations of the processes of the nerve-cells.

## DURA-ARACHNOID AND ARACHNOID CAVITY

**Acute Inflammation.**—This is rare in the dura-arachnoid. The membrane may be involved in an acute process in those cases where disease has penetrated from without. Thus in caries of the spine the sheath of the cord may become involved in the inflammatory processes, and the same sometimes occurs through necrosis of the sacrum, arising from bedsores, and extending to the meninges in the spinal canal.

Let us take for example the case of a lad who had received a blow over the loins; an abscess formed which involved the vertebræ, and the purulent matter entered the spinal canal, so that when he died, three weeks afterwards, the arachnoid cavity was filled with pus, and the inflammatory process had proceeded up to the brain, and there produced also an arachnitis. Several similar cases have occurred from bedsore, where, owing to necrosis of the sacrum, the lower part of the spinal canal was laid open, a purulent inflammation extended along the arachnoid, and also reached the skull. Thus, a man had paraplegia for a month; a bedsore formed and extended frightfully, causing necrosis of the sacrum; the inflammation then reached the meninges, and set up a dura-arachnitis; the arachnoid cavity was filled with puriform lymph. In this way the original spinal disease—softening of the cord—produced indirectly a second and totally different spinal disease; we have now seen several such cases.

**Chronic Inflammation.**—Most of the changes seen in the dura mater are of a chronic character; thus in disease of the spine, of which we have been speaking, after the canal has been reached, some lymph or scrofulous matter is formed on the outer surface of the dura mater, and extends sometimes along it for a considerable distance, and so thick layers of deposit are found covering it, as well as the nerves; the inflammatory process may extend through the membrane, or the tuberculous matter may be found protruding on its inner side; but it is surprising how long the dura mater will resist the passage of inflammation. Unhappily, however, it does not prevent the tuberculous abscess pressing on and destroying the cord, an effect that may be produced when the curvature of the spine is very slight.

In chronic inflammation, not arising in the bone, the medulla and all the membranes are involved together, with local or general adhesions; just as we find in the brain, so in the cord, it is often difficult to say in what structure the morbid process commenced, or whether it has not proceeded in all *pari passu.* In some of our specimens we see the membranes adherent to the cord, and in one the dura mater is ossified, and in some parts forms a complete bony sheath around it; it appeared by the microscope to consist of true osseous structure. In some cases chronic spinal meningitis is evidently syphilitic, coexisting with gummatous disease in the liver, &c.

**Tubercular Meningitis.**—This affects the dura-arachnoid of the cord, although it very rarely indeed, if ever, attacks the cranial dura-arachnoid, and then only in traces; but in the spinal canal we may find in cases of tubercular meningitis the dura-arachnoid crisp, thick, and vascular-looking, and having its inner surface sprinkled with numerous fine, sand-like, tubercular grains. Naturally the spinal dura-arachnoid is very thin ; if laid on fine print we can see to read through it quite plainly. In these cases of tubercular arachnitis it often becomes too opaque for this. We have for a long time drawn attention to this curious pathological difference between the cranial and spinal dura-arachnoid.

**Morbid Growths.**—The tumours that affect the dura-arachnoid mostly proceed from without to attack this membrane. Thus tuberculous disease of the spine, or malignant disease beginning either in the bones or around the spine, penetrates to the canal, and involves the sheath, or even the cord within. Many specimens of this may be seen on our shelves. In one curious and interesting case observed by Mr. Campbell de Morgan a cancer in the anterior part of the base of the skull was associated with cancer in the meninges of the spine, suggesting that the germs of cancer had spread along the subarachnoid space. The spinal growth is often secondary to cancer of the breast, and by involving the nerves constitutes a most painful form of paraplegia.

Virchow gives a remarkable case of *enchondro-lipoma*, which arose in an old soldier, between the bones and dura mater in the dorsal region, and compressed the cord fatally. He thought it might be congenital. Somewhat similar are certain compound mucous and cartilaginous tumours. Thus, we found a tumour congenitally developed in the sacral region attached to the meninges at the lower end of the spinal canal ; in this there were cartilage, bone, brain, ciliated mucous membrane, &c., in short, almost all the tissues of a fœtus. Such tumours are more frequent in the sacral and coccygeal region ; they are generally situated in the middle line. Tumours arising primarily within the compass of the spinal dura mater, or from its substance, are comparatively rare. The few that have been recorded are generally spindle-cell and fibrous growths, of *sarcomatous* nature, which come from the arachnoid face of the dura-arachnoid in the same way as similar growths spring from the arachnoid side of the dura mater of the skull. They are mostly oval and about the size of a hazel nut. We have also met with a *fibroma* and *psammoma*. *Cancerous* growths are occasionally met with, but are not primary. They may be found on the spine, and not on the dura mater, in cases where they are seen between the cranium and dura mater, proving that the disease belongs essentially to the bone.

## PIA ARACHNOID MEMBRANE AND SUBARACHNOID SPACE

We take these together because pathologically they suffer together like the corresponding parts upon the brain.

**Inflammation.** *Acute.*—The superficial or arachnoid membrane is almost evascular, while the blood-vessels are in the deeper membrane lying immediately over the cord; consequently all idiopathic inflammatory exudations take place from the deeper membrane, and are collected beneath the arachnoid. This is analogous to what we said with respect to inflammation of the membranes of the brain, that in this affection the exudation in primary inflammation was not interarachnoid, or between the serous surfaces, as we might have thought by judging of the analogous instances in the other serous cavities of the body, but beneath the serous surface in the subarachnoid space. We must remember, however, that in the spinal as in the cranial membranes a pia-arachnitis may arise from external causes, though a dura-arachnitis does not arise from internal causes. Spontaneous spinal meningitis therefore is the same as pia-arachnitis, and we look for the products of inflammation under the arachnoid surface. What first strikes the eye before opening the membrane is the remarkably irregular look of the cord, owing to the effusion of lymph within the arachnoid space; this space, as we know, can be distended with air into a large sac, and being now occupied irregularly by inflammatory exudation the whole surface of the medulla acquires a very uneven aspect. On cutting the arachnoid the lymph is found beneath as a yellowish-green material, toughish, softer or even puriform; in some cases it is more in the front, and in others on the posterior part of the cord.

It is not often that we meet with acute spinal pia-arachnitis without the cerebral membranes being affected at the same time; we know what free communication exists between the cerebral and spinal arachnoid spaces.

*Cerebro-spinal meningitis; cerebro-spinal fever.*—Acute meningitis of the cord is generally speedily fatal. This of itself would ensure attention to it, but its most serious interest is in respect of its occurrence in the form of the so-called cerebro-spinal fever, which has been epidemic on the Continent, in Dublin, in America, &c., though never to any extent in our own island.

Like other grave fevers it may come down on its victim altogether overwhelmingly, so that he becomes dusky and purpuric, and sinks in a very few days before any very obvious products are formed in the meninges. But when the case is as usual of mitigated yet fatal severity we find it a pia-arachnitis, the lymph lying chiefly on the dorsal surface of the pia mater. Only in the very rarest cases do any products appear in the arachnoid space. The spinal cord itself in this disease is often quite free from change, but sometimes a slight inflammatory formation is traceable along the lines of the medullary rays for some little distance into the medulla.

In a case which occurred here there was a patch of purulent lymph on the surface of the pons and in the Sylvian fissure. The lateral ventricles contained an excess of turbid fluid and lymph, extending some way down the cord. Sometimes lymph has been found the whole length of the cord. The roots of the cerebral nerves are often involved in a most important manner in the inflammatory process.

Our own experience of pia-arachnitis or spontaneous meningitis is limited. We have met with it as a complication of the last stage of heart disease. Dr. Murchison has recorded a case of the same kind. We have twice found it in persons with evident constitutional syphilis, once in a woman with syphilitic rupia and characteristic gumma of the liver ; once also in pyæmia. One thing we would remark here, though the point is rather clinical than pathological, and that is, that the set of symptoms which even in our latest text-books are given as characteristic signs of spinal arachnitis, viz. tetanoid stiffness and pain in movements of the limbs, have not existed in those cases where we have verified the meningitis post mortem. Thus, in one case a boy who had pus filling up his subarachnoid space stood up in his bed restlessly, and got out to micturate only a few hours before his death. In another case the woman was found wandering from bed to bed on the only night she lived after admission to the hospital. In another the limbs were described as "tossed about." Another suffered from languor, heat, thirst, and, finally, convulsions.

*Chronic inflammation.*—The pia-arachnoid is occasionally found thickened and opaque in persons who have long suffered from spinal symptoms. It then is commonly more or less adherent to the dura mater. This appearance, on the whole, is very rare. Most of the chronic spinal diseases implicate the cord proper and not its membranes. It is necessary to note that there are fine adhesions by minute points between the dura- and pia-arachnoid normally present in the cervical region. These we have often found incorrectly described as a pathological state.

A local meningitis confining itself to the cervical region of the spinal cord has been so often met with that Charcot has given a distinct name to it, *hypertrophic cervical pachymeningitis.* The spinal cord for about three inches of its length is firmly united to the membranes around by a quantity of inflammatory material. This is in such quantity and is so dense and circumscribed that the term tumour has sometimes been applied to it. The nerves passing from the cord are also usually involved in this inflammatory mass.

Local gummatous *syphilitic* inflammation when occurring resembles exactly the like gummatous meningitis of the brain. We shall speak of it again under "Medulla."

**Congestion and Apoplexy.**—We have never seen a case of apoplexy of the spinal membranes, but such disease is said occasionally to occur, and gives rise to sudden paraplegia. Blood is frequently found in the spinal membranes as an extension from intracranial hæmorrhages, either traumatic or spontaneous. We have known it produce additional symptoms in such cases. As to congestion we would refer to the remarks made on congestion of the brain. Post-mortem fulness of blood can never prove an active state of congestion, nor have any real pathological importance. Owing to the mode of death, and the body having been long supine, the veins may be found filled with dark blood.

**Morbid Growths.**—Tumours on the pia-arachnoid of the cord are very rare; we have but one specimen composed of wavy fibrous tissue and long nuclei. It is said that *carcinoma* has occurred there, and Virchow remarks that *sarcoma* is more frequent on the spinal than on the cerebral pia mater. Once we met with a curious elongated *cyst*, three inches long by half an inch wide at its widest part, on the dorsal aspect of the lumbar part of the cord, firmly connected with the free surface of the arachnoid, reminding one of the hæmorrhagic cysts in the cranium, but the latter are more connected with the dura mater.

Gowers reported a case of a tumour growing from the pia mater at the end of the cord, and which he called *myo-lipoma*. It was composed of fat, with some fibrous tissue and striated muscular fibre. It was so peculiar in structure that he regarded it as congenital, having its origin in some fœtal structure.

A short time ago a girl was in the hospital with complete paraplegia. There was found after death a *gliomatous* tumour three inches long and one inch broad growing within the membranes at the posterior part of the cervical region and compressing the cord.

*Psammoma* has been found in the membranes of the cord as well as the brain, and, as in the brain, composed of calcareous particles. These are concentric globules formed in the endothelium and calcifying in their centre.

The spinal arachnoid very commonly is the seat of curious *bony* plates; they are very rarely met with on the pia-arachnoid of the brain. They are so common over the cord that often we have found them there in each of five or six bodies successively; half the population at thirty-five have a few. It is a great and a rather common proof of ignorance of the spinal cord to treat them in a post-mortem examination as if they bore on the symptoms of the case.

They are white, smooth, irregular-shaped plates on the arachnoid, seen on opening the dura mater. Their most frequent seat is at the lower and posterior part, especially on the cauda equina. They are white, like porcelain, or, when very small, like grains of split rice. If we try to remove them, they are found firmly united to the membrane all around their circumference; and we notice that though the outer face is smooth the inner is surprising from its extreme roughness due to papillose projections, highest at the centre of the patch. These have long been known by the name of bony plates on account of their hardness; but the correctness of the name has been questioned, because often no bone can be found in them, and they have therefore been called cartilaginous. The tissue of which they are composed is very like bone, but often without lime-salts. We see in it cells with out-branching processes, resembling bone-cells, with more or less of hyaline or fibrillated intervening substance. The branches of the cells often meet, so that when calcification sets in the transformation to bone, with its lacunæ and canaliculi, is immediate; the result, not being, perhaps, perfectly typical bone, yet sufficiently characterised.

As to the significance of these, they were once thought to cause epilepsy, tetanus, &c.; but they are probably innocent, at least, we have often found them largely developed, without any spinal symptoms. Virchow suggests that they rather result from than cause irritation. But the freedom from symptoms is surprising when their size and number, and their roughness towards the cord is considered; we counted thirty-four large plates in one case. They are rarely on the front of the cord. They increase with the age of the individual, but we have found many large ones in a person of twenty-four years.

## MEDULLA SPINALIS

**Myelitis**, or inflammation of the spinal cord, is a general term formerly made to include many affections, which we shall have to describe under distinct names according to the region of the cord involved. At the present time the term is not used, except a large part of the cord is indiscriminately affected by an inflammatory process. According as the white or grey matter is affected so the terms *leucomyelitis* and *poliomyelitis* are sometimes employed. Myelitis is conveniently divided into the *acute* and *chronic* forms; the former is often synonymous with *softening*, whilst the latter, in which the tissues are so often hardened, agrees more or less with *sclerosis*.

*Acute myelitis and softening.*—Although acute myelitis produces softening, the two expressions are not co-extensive, for softening may occur under other circumstances, as from mechanical pressure or the occlusion of an artery by an embolism. Care should be taken not to confound softening with the artificial crushing of the cord, caused by the awkward use of the bone forceps. If the cord be softened at any spot, this is readily appreciable by passing the finger along the surface until the pulpy part is reached, when it sinks in. On making a section this is felt to be softer than other parts of the cord, and the knife tears and hacks up its substance instead of cutting cleanly through it. It might also have become swollen and lost its colour, and the grey matter its definition, appearing darker from small extravasations of blood. If the whole portion is hyperæmic, the term *red-softening* is sometimes given to it as in the brain; or *yellow softening*, when at a later stage it has undergone liquefaction or fatty degeneration. An examination by the microscope shows the breaking up of fibres, and the presence of granule masses and fatty particles. The medullary sheath has apparently at first undergone a change, the myeline coagulated and formed in masses, which have become granular and broken up, so that the nerve puts on various shapes until it is quite destroyed, or leaving for a time the axis-cylinder free. This, however, may become sunken and assume a varicose appearance. In the grey matter the cells become swollen and cloudy, with all their processes irregular. The neuroglia is crowded with numerous nuclei, and the blood-vessels are surrounded with fatty granules. The connective-tissue cells, or Deiter's cells, often become more marked or perhaps more developed. In cases where paralysis arises from pressure, as in disease of the vertebræ, and recovery takes place, in all probability the axis-cylinders have never been affected. In the acute ascending, or Landry's paralysis, no changes whatever have been found in the cord, and therefore it is not an acute myelitis as has been surmised.

*Chronic myelitis.—Sclerosis and grey degeneration.*—Whether all these conditions are the same, as well as their mode of production, is not quite determined. Some have used the term simple grey degeneration where specially localised parts of the cord have been affected, leaving the word inflammation for the cases where the changes have been more extensive. In all cases this degeneration is recognised by the greyish change in the medullary structure, and by this being harder; therefore the meaning of the name sclerosis. The microscope shows it to be due to the production of a quantity of fibrillated connective tissue spread amongst and encroaching on the nerve-fibres. It shows also masses or tufts of fibrillated tissue proceeding from cells of the connective-tissue type. The nerve-fibres in their sheath become fused with the new material until they altogether disappear, but often, although welded into a felt-like mass, the axis-cylinder remains intact for a considerable time. In early stages, before the degeneration is seen by the naked eye, staining fluids like carmine will display the new tissue.

It is usually considered that the source of the new material is the neuroglia; a still further question being open whether this binding material is simply granular or fibrillated. It is generally believed that the latter is its true nature and of a connective-tissue character. From the cells in this neuroglia the new material is derived which presently swallows up the true nerve-tissue. It is still, however, held by some good authorities that the nerve-fibrils themselves are the source of the new material, and that the morbid process primarily begins in them. A "parenchymatous sclerosis" is therefore spoken of as distinct from an "interstitial sclerosis."

Charcot asks which is the primary seat of the lesion. Is it the neuroglia or the nerve element? He thinks the latter, or that parenchymatous irritation is the initial fact. He thinks, however, that in disseminated sclerosis the disease may begin as a hyperplasia of reticulated fibres of neuroglia followed by a degenerative atrophy of the nerve elements. Buzzard and Rindfleisch think that the sclerotic changes may sometimes begin in the blood-vessels. There are, however, good histologists who hold that in all forms of sclerosis the first stage is an atrophy of the nerve and the changes in the neuroglia secondary,—that while the disease is isolated, the axis-cylinders remain intact. When destroyed, the sclerosis becomes diffused. In whatever way it comes about, the result is a hyperplasia of connective tissue, which hardens and contracts, the nerve-fibres and cells being involved and finally destroyed, the true connective-tissue cells, or Deiter's cells, being much increased in size and number. In the midst colloid or so-called amyloid bodies may be found.

In considering the causes of grey degeneration or sclerosis a very important element cannot be overlooked. It is found that, if any injury takes place to the fibres of the cord in any region, degeneration will commence and continue along the course of those fibres, as if their nutrition depended upon the integrity of the whole tract. In the same way as when a nerve is divided a degeneration will occur along its course, as if some nutrient influence had been cut off. In these cases the term degeneration is used instead of sclerosis, although the result in both is much the same; the exciting causes being apparently different, in one case, progressing from a process where a lesion has occurred, in the other occurring quite independently as must be the case when taking place simultaneously in a number of isolated patches.

*Grey degeneration or sclerosis* is mostly met with in the medullary portions of the cord, and may be spoken of as primary or secondary.

A primary sclerosis of the cord is met with in the lateral or pyramidal columns under two forms, continuously throughout them, or in isolated patches. It has been met with involving the whole of the lateral tracts throughout the cord and affecting necessarily both sides; but not many instances of this primary affection, however, have been met with. In some it has been limited to portions of the lateral tract; in others it has involved the whole, and sometimes included the cornua and other portions of the grey matter. It is this primary sclerosis of the lateral column which is supposed to exist in *spastic paralysis*. A much more marked variety of sclerosis of the lateral columns is the disseminated form. This is found in distinct or insular patches throughout the cord; they are of various sizes, and sometimes two or three are seen on a section. They may be met with throughout the whole length of the cord, and extending upwards through the pons and crura to the brain. In the latter they are seen as grey patches in the white substance, and in some exceptional instances involving the cortex of the brain or the grey ganglia within. This is the disease found in *rhythmical paralysis*. In one of the cases described by Dr. Goodhart in 1876 the whole brain and cord were hard and rigid. A section of the brain showed patches of a circular outline, about the size of a silver penny and of a grey colour. Some were very small like points. They were seen on the roof of the lateral ventricle, in the fourth ventricle, and in the medulla and pons, while the cerebellum escaped. The grey matter was unaffected, except where slightly encroached upon in one or two places. The morbid parts were darker and more translucent than the healthy structure. In another case Dr. Hale

White showed how the sclerosis corresponded with the rays of neuroglia passing in from the surface. The microscope showed in the midst of the new connective tissue many axis-cylinders still remaining. Another primary sclerosis is found in the *posterior column* in connection with locomotor ataxia. Those who have had especial opportunities of making observations maintain that the primary sclerosis takes place in the posterior root column, or the column of Burdach. In some simple cases no other part has been affected; very often, however, the internal strand, or column of Goll, becomes involved, and then as the disease passes upwards the whole of the posterior column is found involved, most markedly in the lumbar and dorsal regions, and ceasing at the fourth ventricle. In some cases the posterior cornua have been involved together with the roots of the nerves and also the cerebellar tract. It has been maintained from this fact and a consideration of the symptoms of anæsthesia and pain that the disease begins in the posterior roots, and proceeds inwards to the columns of the cord. In these cases, as in others, it is still a disputed point whether the process should be called one of inflammation or degeneration.

*Friedreich's disease, or congenital ataxia.*—The posterior columns have been found sclerosed, more especially in the lumbar region. In a case carefully examined here by Dr. Pitt there was complete sclerosis of the columns of Goll throughout the cord with various degrees of sclerosis of Burdach's column, and of parts of pyramidal tract and of cerebellar tracts. The shrinking of the cord at the cervical and lumbar enlargements was very noticeable.

A primary disease may begin also in the grey matter. This, if inflammatory, is called *poliomyelitis.* The best known form is that occurring in *infantile paralysis;* the case where after a short febrile attack one or more of the limbs of the child are found paralysed and afterwards become atrophied. An opportunity of seeing this inflammatory process was given to Dr. C. Turner, who found the grey matter reddened and otherwise altered in colour, and by the microscope it displayed abundance of leucocytes, the stellate cells being swollen and the fibres altered in form.

Dr. Angel Money also had an opportunity of observing an acute case. He found in the lumbar enlargement a red area occupying each anterior cornu. This had at its periphery a translucent border, and the microscope showed a distension and thrombosis of the vessels; it was infiltrated with leucocytes, and there was an absence of multipolar cells. He believed that the grey matter was more affected than the white because less well supplied with blood, and that the lumbar part of the cord generally was less supplied than the cord above. He agreed with the author of the Gulstonian Lectures (Moxon) that this was due to the spinal arteries entering the cervical and upper dorsal arteries almost horizontally, whilst those in the dorsal and lumbar regions have to ascend a considerable distance before they reach the cord.

In cases of long-standing infantile paralysis the anterior cornua are found atrophied opposite to the limbs whose nerves they supply. The ganglionic cells may be absent or malformed, and the axis-cylinders have generally disappeared; the region of the anterior cornu has a felt-like appearance. The antero-lateral column below may be more dense from the presence of connective tissue, and the anterior roots of the nerves may be wasted.

In spinal paralysis with wasting a similar atrophy of the anterior cornua has been found in association with disease of the lateral columns. The case, however, is different when the muscular atrophy has been the earliest symptom, for then, even if changes in the cord be found, they are generally regarded as secondary. When, however, the paralysis is primarily indicative of disease of the lateral columns and a wasting follows, we expect to find an atrophy of the anterior cornua as well as chronic myelitis of the motor tracts. To this condition the name *amyotrophic lateral sclerosis* has been given by Charcot. Dr. Coxwell described a case of this kind at the Pathological Society. The limbs were wasted and contracted, especially the arms and hands. The lateral columns showed by staining that the whole length of the pyramidal tract was diseased as well as the direct columns of Türck. There was also atrophy of the multipolar cells of the anterior cornua; they were withered and altered in shape. Slight changes were also found in the grey matter as high as the fourth ventricle.

In the analogous case of *bulbar paralysis*, where there is wasting of the muscles, changes are found in the grey matter along the raphé of the fourth ventricle, and involving the grey centres of the seventh, eighth, and ninth nerves. The nerve-roots are also atrophied. We might here allude also to—

*Progressive muscular atrophy.*—It has long been a question whether this disease is a neuropathia or a myopathia; that is, whether the disease begins in the nerve-centres, or whether it commences in the muscle and periphery of the nerve, and travels backwards to the cord. In fatal cases the muscular tissue has become atrophied, together with the motor nerves, the anterior spinal roots being markedly smaller; and at the same time the spinal cord may be visibly shrunken on its anterior aspect. All these conditions were well marked in a case which occurred here, and, moreover, Dr. Thompson Dickson found, on subsequently examining the cord, a most marked atrophy of the ganglionic cells in the anterior cornu. Some have described the production of an increased fibrous tissue in the lateral columns, or a fibrosis of the neuroglia.

Resembling this disease in its final effects is—

*Lead paralysis.*—Observers are not yet agreed as to the immediate cause of this disease. Some have maintained that there is a primary myositis followed by a neuritis, which then proceeds to the cord; others have believed they have found evidence of an anterior poliomyelitis by an altered form and size of the grey cells of the anterior cornu.

*Alcoholic paralysis.*—Nothing distinctive has yet been found, and this may be taken in connection with the fact that in some of the most severe forms of paralysis arising from alcohol, in which atrophy and contraction have taken place, a complete recovery has occurred. There is a very prevalent opinion at the present time that the affection is not a central one, but a neuritis.

*Pseudo-hypertrophic muscular paralysis.*—Nothing very characteristic has yet been discovered in the nerve-centres, although some authors have described slight degenerative changes. The muscles are of a yellowish-white colour, and have undergone a translucent, waxy, or granular degeneration.

*Paralysis agitans.*—Here again nothing has yet been positively made out in the nervous structure, but Dr. Cayley reported a case in which he believed there was an increased amount of connective tissue in the medullary matter of the cord.

The *secondary degeneration*, although much of the same anatomical nature, appears to originate in a local lesion, and then proceeds upwards or downwards according to the anatomical and physiological course of the fibres—downwards in the motor tract, and upwards in the sensory. It would seem from this that they owe their nutrition to a distant source, since degeneration speedily occurs in the fibres of the cord after their severance, just as it does in a peripheral nerve. The fact of the degeneration following certain courses is taken as an evidence that these strands are distinct. A further confirmation of this has been made by a study of their embryological development, by which method it is seen that the strands of the cord grow at different rates, so that then separation from one another is easy (Flechsig).

*Descending degeneration.*—One of the most interesting of these degenerations is that which is seen in the lateral or pyramidal tract. This commences in the brain in connection with the convolutions which form the motor region, such as the ascending frontal and parietal, bordering on the fissure of Rolando. Now, the fibres from these convolutions pass down by means of the corona radiata between the caudate and lenticular nuclei, and afterwards between the latter and the optic thalamus; here the bundle of fibres is known as the internal capsule. These motor fibres then pass through the crura and pons to the medulla, where they bifurcate; some passing to the opposite side, and others passing down on the same side of the cord. This tract may undergo the degenerative process, so as to involve nearly its whole length. In these cases the grey matter is not touched, and therefore, the motor nerves escaping, there is no atrophy of the muscles.

This degeneration of the motor tract may be traced, we say, from the brain to the peduncle, and then through the pons. The former, or crus, may be found in marked cases flattened, and when cut into may show a grey band running through its middle and towards its lower part or crustum. In the pons Varolii the degenerative tract is more difficult to trace; it passes on, however, to the medulla, and where it crosses at the pyramids the altered fibres are clearly visible. The section of the cord below this shows that the whole tract is not involved, but merely a portion lying near the posterior cornu. This portion is usually triangular in shape, and has in all probability involved the long descending or cerebro-spinal fibres, leaving untouched the shorter transverse motor fibres. It is in the primary affection of the cord that so large a part of the lateral tract is involved. Besides this crossed degeneration there may be often seen a slighter degeneration, affecting the non-decussating fibres which run along the sides of the anterior fissure known as the column of Türck. This degeneration of the motor columns becomes less and less as it passes downwards, and is rarely traceable beyond the dorsal region. It keeps its own special course, and does not tend to encroach on the neighbouring structures as the grey matter and nerves. It is a remarkable fact that although the disease may begin on one side of the brain, and consequently involve one motor tract, yet after the bifurcation at the pyramids degeneration may pass down both sides of the cord. Hadden had a case where, from cerebral hæmorrhage on one side, degeneration occurred and proceeded downwards on both sides of the cord.

Some very marked forms of descending degeneration commencing in the brain are seen in congenital cases of hemiplegia, with contraction or atrophy of the limbs. In such cases, of which a good example is given by Dr. Taylor in the 'Guy's Hosp. Rep.,' the brain was atrophied on one side (perhaps the result of a clot in infancy), and degeneration had passed down the pyramidal tracts and down the spinal cord as before mentioned, the posterior part of the cord being the part selected. In a case of atrophy of one side of the brain from embolism, Dr. Coats traced degeneration down the whole length of the cord. The crus cerebri was visibly shrunken, and also one of the pyramids. The degeneration continued down the lateral columns, occupying from a third to half of the surface of a section. In the dorsal region the cord was less affected, and in the lumbar region still less. In Dr. Taylor's case there was observed to be some wasting of the lobe of the cerebellum on the opposite side to the pyramidal tract.

This observer also had another interesting case. It was that of a child, aged six years, who fell on to the back of its head about a year before its death, and from that time onwards had symptoms of chronic cerebro-spinal disease. The post-mortem showed the posterior half of the corpus callosum hardened, as well as the posterior part of the brain, with a portion of the temporo-sphenoidal lobe and some of the medullary matter around the central ganglia. A hardening also as a secondary process had taken place in the crus cerebri, pons, medulla and cord. The pons showed distinct tracts of translucent material on both sides, as also did the crura in their lower part or crusta. The degenerative tracts united in the medulla, and then again separated at the decussation, to be seen further running down the lateral columns.

*Ascending degeneration.*—This may be seen passing from below upwards, and observable more particularly in the posterior columns which run along the fissure, known as the columns of Goll. It has already been said that in the primary sclerosis of ataxia the external column or root-zones are alone affected, but often the internal, or those of Goll, become subsequently implicated, so that the morbid process proceeds upwards in the whole of the posterior column. If a lesion of any kind should affect these posterior columns it may start a degenerative process therein, which will slowly proceed upwards. Some few cases tend to show that degeneration may begin in the nerves of a limb, then affect the cord and travel upwards. These consecutive degenerations are well shown in a specimen in the Museum put up by Dr. Taylor, where the cord was injured in the dorsal region; an inflammatory process was set up, and degeneration was seen proceeding in both directions upwards and downwards, upwards in the course of the columns of Goll, and downwards in the lateral tracts.

Another tract of ascending degeneration has often been noticed when any inflammatory affection has commenced in the dorsal region, and more especially in the column of Clark, viz. a tract on the outside of the lateral columns. This degenerative thin layer of new grey tissue may be traced upwards as far as the restiform bodies, and, as it is supposed that the fibres affected are those which pass into the cerebellum, this layer has been called the *cerebellar tract.* It has not been traced lower than the dorsal region.

If the degenerative process in the cord depends upon severance of the fibres of some nutritive centre, it would be necessary to believe that the motor tract received in the first place some nutritive influence from the motor region of the brain, and in the same way that the fibres in ascending degeneration possessed some source of life in the origins of not only the posterior columns but of the cerebellar tracts.

A very interesting observation was made by Drs. Hadden and Gowers about three years ago in a case of ascending degeneration. They found, besides the parts usually affected, another tract of grey degenerative tissue ascending in the lateral columns. This was situated on the outer side of the anterior cornua, and its section was of a comma shape. The existence of this ascending tract in the lateral region has since been verified by others.

It has already been said that from a pathological point of view it seems important to distinguish between those chronic changes in the cord which are primary or inflammatory, and those which are due to secondary degeneration, yet from an anatomical examination of the morbid change it is often difficult to do so. To call the one sclerosis and the other grey degeneration, making the former depend upon changes in the neuroglia and the other in the nerve element, is not very satisfactorily proved, much less so since there are reasons to think that the primary change may occur sometimes in the blood-vessels. This difficulty prevents us at present from affirming which are the primary and which the secondary changes in the cord, when it is found much diseased in illnesses of long duration. Thus, it has already been said that the posterior root columns are those which are primarily affected in tabes, and yet very often we find the whole of the posterior columns diseased. It is then suggested that the columns of Goll were secondarily involved. In the same way a large number of cases have now been recorded where the lateral columns have been affected in common with the posterior.

*Syphilitic disease of the spinal cord.*—The cord is not found to be affected so often as the brain, but we have met with several cases of meningitis of a syphilitic nature where the membranes were adherent to the medulla by gummatous material, and more rarely, we have met with gummatous tumours within the substance of the cord itself. The first case which came under our notice was one where a hard deposit was found in the lumbar region of the spinal cord. This was nearly an inch in length, and had apparently grown up along the posterior roots of the nerves. It produced a paraplegia, and syphilitic deposits were found in other parts of the body.* In another case, of a young man, there were several small deposits in the lateral and posterior columns. One in the lumbar region occupied half the lateral column. This was composed of fibrillated tissue with nuclei. There was deposit in other organs.† As a meningitis similar to the affection of the membranes of the brain it is more common. Thus, in the case of complete paraplegia in a young woman, the cervical region of the cord was closely enveloped in a gummatous material uniting it firmly to the membranes around. Occasionally, as in the brain, the spinal symptoms are brought about by softening from syphilitic disease of the vessels. Thus, a lad who still had upon him the external symptoms of syphilis became rapidly paraplegic. He lay in bed several weeks with the usual symptoms until he died. On examination of the cord no growth or other morbid appearance was seen, but there was a spot of softening in the dorsal region. A section of this showed broken-up tissue and the grey matter quite confused; when some of this was placed between two pieces of glass the blood-vessels were seen to be very much thickened and opaque, contrasting strongly with the smaller, thin-walled, vessels of the healthy portions of the cord.

In the brain the diseased vessels have sometimes led to thrombosis.

*Sanguineous apoplexy of cord.*—It is a great question whether a primary effusion of blood ever occurs in a healthy cord from the rupture of a blood-vessel, as is so common in the brain; when effusion does occur it is the result of a previous softening in the cord itself. For example, a man had been ill for many months with paraplegia when suddenly he became much worse and died. At the lower part of the cord, where softening existed, there was a recent clot of blood. In another case of paraplegia, of two years' duration, the man suddenly became worse and died. In this case the membranes were found full of blood which had proceeded from a softened spot in the dorsal region. The Museum contains a specimen where the membranes are full of blood, but the source of this was the rupture of a carotid aneurysm. Dr. Cheadle described the case of a child aged four years where hæmorrhage was found in the grey matter of the spinal cord; more especially in the cervical and lumbar enlargements. The child had been ill ten days. It was found on examination that abundance of change had occurred in the cord by the production of leucocytes, &c., and therefore the case was one of acute myelitis. It probably was of the same nature as infantile paralysis. In various blood diseases small hæmorrhages may occur in the cord, and so be one of the causes of death.

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* 'Guy's Hospital Reports,' Series 3, vol. ix.

† Ibid., Series 3, vol. xvi.

In a case reported by Dr. Jackson, of Notting Hill, of a young lady who was seized rather suddenly with paralysis of the arms and chest from effusion around the spinal cord, the source of the blood was in the cerebellum, a seat of apoplexy before observed in young people. She was fourteen years of age, and after premonitory symptoms of weakness and strange feelings in the arms which lasted two days, she became quite paralysed. In three days more she died, and a clot of blood was found in the cerebellum, and a coagulum surrounding the cervical portion of the cord. Occasionally hæmorrhage is the principal cause of death from injury (*hæmatomyelia*).

*Embolism of the spinal cord.*—We are not aware that we have met with a case of this kind, but probably such sometimes occur. A case was reported from Vienna of a boy aged sixteen who was suddenly seized with pain in his back and legs, and shortly afterwards complete paraplegia came on. He lingered on for six weeks with the usual symptoms when he died. The lumbar region of the cord was soft and the vessels filled with fibrin. Infarcts were found in the spleen and kidneys, and vegetations on the mitral valve.

*Hydromyelocele.—Hydromyelus.—Syringomyelus*—In many forms of paraplegia of obscure origin the spinal cord has been found to contain cavities within it. Sometimes, however, they are met with in children where no symptoms have existed. These cavities are of two kinds, they are sometimes no more than the central canal immensely dilated, and at other times the cavities are evidently of recent formation and pathological, and due to the breaking down of some inflammatory product or neoplasm; for the thickening of the tissues and hæmorrhages are all evidences of myelitis. Writers seem inclined to use the term hydromyelus for the congenital enlargements and syringomyelus for the pathological. Although the irregularity of the cavities clearly show the mode of formation, it is remarkable that they should usually be found in the posterior columns and often lined with a cylindrical epithelium. The explanation of this is that at an early age the canal extends as a fissure between the posterior columns to the periphery, and becomes enclosed by the transvere development of the posterior column. This fissure becoming partly obstructed, there remains a cavity in the posterior columns as a kind of diverticulum. Many examples have now been published where cavities and fissures were found in the midst of a soft gliomatous material. This was seen to be undergoing disintegration, when examined by the microscope. Westphal accommodates the pathological view with that of distension of the natural central canal by remarking that behind the commissure the central canal proceeds backwards, and the diverticulum from it is of a T shape. An obliteration of this at the point where the lines join would leave a cavity in the half of the cord which may then dilate.

Sir W. Gull described, many years ago in the 'Guy's Hosp. Rep.,' the case of a man who had progressive muscular atrophy, and in whose cord the central canal was greatly dilated from the fifth cervical to the fourth dorsal. Dr. Whipham had a somewhat similar case of progressive paralysis, but in his the canal was a recent formation as the natural one could still be traced throughout. The upper part of the cord was enlarged by neoplastic material, which had softened, and dilated into several canals with smooth walls. Dr. Norman Moore reported the case of a woman who had heen partially paralysed in the arms for two years, when there was found a cavity extending the whole length of the cord, and large enough to admit the little finger. The ventricles of the brain were also distended with fluid. Dr. F. Taylor reported the case of a child, who, at the age of five months, had a cerebral attack, after which the head became much enlarged, and the arm paralysed. After the child's death the brain was found to contain a pint and a half of fluid, the spinal canal was flaccid, and a section showed it to be hollow. The cavity extended from the cervical to the lumbar region through the whole length of the cord, and in places it was found to be lined by epithelium. Dr. Taylor thought that the cavity was adventitious, for the small central canal could still be seen running down the cord in front of the newly-formed one. Dr. Taylor also had the case where there was a mass of inflammatory matter involving the cord in the dorsal region. A cavity was found running down the cord, and this was clearly adventitious, for the normal central canal was seen passing down behind it. Dr. C. Turner has suggested that the cavities might arise from the distension of perivascular spaces; which would be analogous to effusion into the ventricles of the brain from serous exudation of the blood-vessels.

Having spoken of the cases of spinal diseases which are essentially primary, there still remain to be described cases where the morbid changes are but slightly marked, and therefore placed doubtfully in the category of purely nervous affections.

*Chorea.*—In this disorder the most marked and almost universal morbid appearance is found in the heart, as shown by a row of bead-like vegetations on the mitral and occasionally on the aortic valves. We have never found emboli in the cerebral vessels, but in the spinal cord Dr. Dickinson, who has made a careful study of chorea, has found changes as well as in the corpus striatum. These consist of erosions and hæmorrhages visible to the naked eye; also of other changes of long standing, shown by atrophy of the tissue and the presence of hæmatoidin crystals. These changes occurred in connection with a dilatation of the smaller vessels in the substance of the medulla. His conclusion therefore as to the nature of chorea is that it is due to a hyperæmia of the vessels arising from causes mainly of two kinds—one being rheumatic, and the other acting more directly on the nervous system by mechanical or mental shock.

*Tetanus.*—In this disease the body presents no peculiarities. There is rigor mortis, as in other subjects, but sometimes the muscles are found lacerated. The heart and lungs often show the cause of death by asphyxia through spasm of the chest, but there is no rule in this matter.

On examination of the spinal cord very good observers have found changes in the medulla very closely resembling those described in chorea, viz. great distension of the blood-vessels with numerous granules around them or even hæmorrhages, and sometimes even small cavities from the wasting of the tissue. Dr. Carrington has confirmed these observations, and in one case found the grey matter of the whole length of the cervical enlargement containing a cavity, and in the lumbar enlargement there was a similar cavity. The microscope showed degenerated fibres and cells, with dilated vessels, interstitial exudation, and other abnormal appearances about the central canal. It has been stated that micrococci have been found in some of the tissues in tetanus.

*Hydrophobia.*—Owing to the mode of death, the various organs are found congested. The pharynx has the mucous membrane swollen and covered with secretion; other glands enlarged, as well as those at the back of the tongue.

Drs. Coats and Clifford Allbutt have found changes in the spinal cord in connection with the blood-vessels. Crowded round them was a collection of leucocytes, with slight extravasations. Some have found similar changes in the brain.

Ross had also shown an inflammatory process around the vessels, with a perivascular exudation; there was then an infiltration of the tissues, with a degeneration of the nerve elements in the upper part of the cord. Gowers obtained similar results, finding distension of blood-vessels, and a collection of cells which he called miliary abscesses. He remarks on the intensity and the locality of the changes being remarkable, as involving the nuclei of the hypoglossal, pneumogastric, and glossopharyngeal nerves.

Dr. Pye-Smith examined the spinal cord of a dog that had died of rabies, and he found marked changes in the medulla which he called inflammatory. There was dilatation of blood-vessels, with some hæmorrhage around them, and these were mostly found on the floor of the fourth ventricle. In the neighbourhood there were also vascular changes and dilatations. This description would point to hydrophobia as an inflammation of the medulla oblongata.

**Morbid Growths.**—*Tubercle.*—The most frequent growth in the spinal cord is a form of tubercle, which has all the characters of tubercle of the brain, forming a large mass of the colour of milk of sulphur in the tissue of the cord. In all the examples we have seen, the tubercle commenced in the grey matter of one side, and expanding in the tissue around, left only a thin superficial layer of cord substance unaffected; it is worthy of notice that there was scarcely a trace or sign of the presence of the tubercle in the outside view of the cord, so that it would be easily overlooked unless the whole cord were sliced carefully. Its microscopic structure showed in one instance most plainly an extension of the lymphoid tubercle all along the sheaths of the vessels in the cord. Another point of interest in this case was the limitation of the tubercular disease to that zone of the body which corresponded to the tubercle in the cord. The tubercle was in the upper part of the lumbar bulb, and there was slight tubercular affection of the peritoneum of the middle of the abdomen with slight tubercular disease of the mesenteric glands. In this case death was caused by the spinal tubercle only; it is not then correct to say that tubercle of the spinal cord only occurs in cases of advanced tubercular disease. It may be primary; indeed, in this nodular form we believe it is always primary.

*Tumours* of ordinary character arising in the cord as distinct from the membranes are comparatively rare. They generally reach the cord from without. *Carcinoma*, *sarcoma*, and *glioma* have, however, been met with. We have already mentioned under "Spine" *exostosis* of the vertebræ, and alluded to Key's conjecture that they might press upon the cord. Gowers has related a distinct case of the kind. Dr. C. Turner had a case of *lipoma*,—a mass of fat contained in a tender fibrous matrix.

We have seen a section of a most remarkable growth that was formed as a swelling in the dorsal aspect of the lumbar bulb. It occurred in a woman, æt. 33, under the observation of Dr. Seguin, an American physician; the woman died of pressure on the cord by a meningeal sarcoma in the cervical region; a section of the lumbar growth was sent to Sir. W. Gull, by whose kindness we saw it. The swelling consists of a partial repetition of the cord in continuity with its proper texture, so that there is lengthwise another smaller cord which is within the pia mater, and has its own white matter, grey horns, nerve-cells, and commissures; it appeared to us to be a congenital malformation.

**Hydatids.**—Rokitansky speaks of cysticerci as having occurred repeatedly in the cord, but the acephalocyst hydatids outside. We met with a case of crushing of the cord by multilocular hydatids, extending from the spine, and growing into the canal; they did not pierce the dura mater.

**Injury.**—Cases of recovery from injury of the spinal marrow are recorded, yet no new formation of cord tissue is known to occur, so that after grave injuries paralysis is permanent. In fractures or dislocations of the spine the cord within is often frightfully injured, as may be seen in our preparations. The cord may be torn through, and the ends forced more than an inch apart, being connected by lacerated membranes, or the cord may be pierced by fragments, or burst, or, in many cases, the membranes are unaffected, but the mischief is caused to the medulla within by stretching and contusion, whereby the structure is softened into a pulp, and is red from effused blood. Sometimes we find the outside of the cord little injured, but the interior grey substance softened; this arises from the softness and vascularity which allows the blood to be more readily effused into it, and even to proceed some way downwards along its centre. We have seen inflammation and hæmorrhagic changes extend several inches beyond the seat of injury. Death occurs in these cases not so much from the amount of injury produced as from the particular spot affected; thus, the instances of recovery from fractured spine are those where the lower dorsal or lumbar vertebræ are injured; where immediately fatal, the injury is high up in the neck, above the fifth vertebra; and in the more usual class of cases, where death occurs in one, two, or three days, the injury is at the lower cervical or upper dorsal region, so that the ribs being paralysed, and the respiration only effected by the diaphragm, the lungs become gorged, and death sooner or later inevitably results. Possibly, where a violent blow has occurred on the neck, without any displacement of the bones, contusion of the cord may have arisen from concussion.

## NERVES

**Atrophy** of the nerves arises from pressure, or is a mere accompaniment of a general atrophy of the part of the body which they supply; thus we find the recurrent laryngeal nerve wasted and almost destroyed from the pressure of an aneurysm, and in cases of dislocation of the arm, the circumflex and other nerves may have been injured, so as to cause paralysis. The same may occur through the use of ill-fitting crutches. A spontaneous or *idiopathic atrophy* of the spinal nerves has been found in cases of progressive muscular atrophy. This, however, does not determine whether the muscle or the nerve is first involved. In infantile paralysis the nerves are wasted; the healthy fibres remaining are probably sensory.

*Inflammation.*—It is seldom that an opportunity occurs to see an inflamed nerve; but, if so, this is seen to be swollen, red, and infiltrated with leucocytes like other inflamed tissues. It has been called *peripheral* or *interstitial neuritis*, according as the sheath or the internal structure is affected. It is probably not uncommon, as, for example, in cases where acute pain is felt for some days or weeks in a limb, followed by weakness or atrophy of the muscles. Also as a more chronic affection in tabes and alcoholic paralysis. In the latter disease there is often acute suffering in the legs, accompanied by altered sensation and terminating in contraction and atrophy of the muscles. There is now a growing belief that this and some other forms of paraplegia are not due to disease of the spinal cord but to a neuritis, and they are in consequence called peripheral paralysis. Of this kind may be the tropical disease called "beri-beri." There is much pain and weakness of the legs, suggesting an affection of the nerves. In those cases where all the nerves of a limb are affected the term *multiple neuritis* is used. In anæsthetic leprosy and some other diseases the nerves may be found chronically hardened or thickened.

*Injury and reparation.*—If a nerve is divided or injured the medullary sheath soon undergoes a change and is converted into drops of myeline; afterwards the axis-cylinder is broken up and finally disappears, the sheath remaining interwoven with the new connective tissue. If the anterior and posterior roots of the nerves are divided, it is generally agreed, as Waller first showed, that as regards the anterior the distal part degenerates; and as regards the posterior roots, it is the proximate portion which decays, showing that the nutrition of these nerves depends upon the grey matter of the cord and the posterior ganglia respectively. This result, however, is not quite so exact as has been stated. When an ordinary nerve is divided there is little doubt, if the ends are brought together, that it will unite at once by what is called the first intention, judging from the speedy restoration of function after the wound has healed. In some cases, however, the function does not return for weeks, showing that it is necessary for a true regenerative process to take place. Ranvier explains that, under these circumstances, the nerve at the proximal end swells, and that a number of new fibres are formed within the sheath of Schwann. At the peripheral end a mass of granulations form, into which the new axis-cylinders from the proximate end pass and enter the nerve-sheaths. He says that the peripheral end does not regenerate, but only receives the nerve-fibres from the proximal end. Other observers, however, maintain that regeneration also takes place at the peripheral end; that axis-cylinders may there form also with myeline around them, and that the nuclei of the sheath are the agents of new growth and repair. All are agreed that when a nerve is divided the usual consequence is for a degeneration to finally take place at the peripheral end; but this is not incompatible with the belief that there is an attempt at repair in the first instance. Mr. Bowlby, in his lectures at the College of Surgeons, said that in the repair of nerves the nuclei of the sheath of Schwann formed the new axis-cylinder, and that the myelin is subsequently formed around them; and that although the distal end after injury tends to degenerate, it does not immediately do so, but on the other hand makes an effort to regenerate without any connection with the proximal end.

**Morbid Growths.**—The most marked changes in the nerves are of the character of tumour; such diseases of nerve are called *neuromata.* They are true or false, according as they are composed of nerve-fibre or connective tissue. They are often met with as *bulbous ends* of nerves, after amputation of the limbs. These are generally composed of fibrous tissue, with a few nerve elements. Dr. Hale White found in one case coiled-up nerves held together by fibrous tissue, but the nerves were degenerated. Neuromata made up of pure nerve tissue are rare. The term chronic inflammation, however, might be employed in some examples, as in the case of the nerves from the upper and lower extremities of a woman who died lately in the hospital, in which nearly every one is enlarged by an adventitious deposit to three times its natural size; this consists of a simple fibre tissue, infiltrated among the fibrillæ, so that they are firmly united together and with difficulty separated; in some portions a large mass is connected with a fasciculus, so that a tumour is produced, forming a nodule on the nerve, the other fasciculi passing over it. In other parts the fibre tissue is diffused amongst the nerves, so that, on dissection, they are merely found separated by the new material.

In another specimen in the Museum all the nerves of the body, including the pneumogastric, had large tumours upon them, one of these being the size of an egg. In one case the nerves of the arm were alone affected. The complaint is not a painful one, and must not be confounded with painful subcutaneous tumours, consisting of nodules of fibre tissue containing fibrillæ of sensitive nerves. Sometimes one nerve may be affected, and if this belong to an important part, like some intracranial nerves, may lead to death. There is reason to believe that neuroma, in many instances, has a syphilitic origin, the adventitious material being of the same kind as that which forms in syphilitic nodes, &c. Dr. Barlow exhibited the brain of an infant at the Pathological Society affected with syphilis, in which the cranial nerves were much enlarged. There was also another case exhibited in which one optic nerve was much larger than the other, and so also were the third and sixth nerves. Occasionally the tumour softens in its interior, and a cyst may be found within; into this hæmorrhage may occur, and thus several examples of blood-cysts on the brachial nerves have been recorded. We have met with destruction of the facial nerve in the canal of Fallopius by effusion of blood into it.

Some examples of *myxoma* of the nerves have been recorded, and some of these have proved malignant in their nature. Myxoma appears to be the most frequent of the single tumours of nerves, but some cases of *sarcoma* are recorded. We lately had in the hospital the case of a girl where two tumours were found at the base of the brain, and which seemed to spring from the seventh nerves.

The various forms of *cancer* often destroy nerves in their neighbourhood, but do not often spread in their tissue. In one specimen, however, we may see the nerves of the brachial plexus enlarged by cancerous deposits, which also continue along their branches. The invasion of nerve-roots along the spine in cancer of the vertebræ is apt to produce a most painful form of paraplegia.

# DISEASES OF THE ORGANS OF SPECIAL SENSE

## EYE

In the eye the several coats are composed of structures which have diseases like to the diseases of the same structures elsewhere; thus the diseases of the conjunctiva are those of mucous membranes; the diseases of the sclerotic are those of fibrous tissue; the diseases of the choroid are those of the pigmented membranes of the brain; the diseases of the retina are the diseases of brain, of which the retina is originally a bud.

Hence it is convenient to consider the diseases of the eye as to the coats they affect; we can only give a cursory summary of them, and refer for further details to special books on the subject.

**Conjunctiva.**—In this we meet with the usual inflammations of mucous membranes. Indeed, these inflammations are, perhaps, best studied in the conjunctiva, where their varieties and peculiarities are most easily recognised. We often see a simple *catarrhal* conjunctivitis or "ophthalmia" through exposure to cold or other irritation; in this there are only physiological or functional changes, including great active congestion, and production of mucus rendered watery by the increased secretion of the lachrymal gland. *Purulent* ophthalmia is rarer and graver; in it the epithelial cells have outwandered pus-cells between and among them; the mucous membrane itself is greatly swollen, and may be full of pus-cells; and the cornea, iris, &c., may be attacked so that sight is destroyed. A variety of this, with *diphtheritic* membranous exudation, is described.

*Herpetic* conjunctivitis appears in the form of little red-based elevations near the margin of the cornea or the sclerotic; *strumous*, with specks on the cornea and great irritability of the eye; and *granular*, where after long-continued irritation a papillose condition arises on the mucous surface of the lids. *Pterygium* is the name given to triangular-shaped vascular thickenings, which extend from the margin of the cornea, expanding outward, generally towards the semilunar folds.

The conjunctiva is subject to *pigment* patches such as are natural in the dark races; little yellowish collections of *fat* may form under it in the exposed part in elderly people. *Nœvus* is found on it, also small *dermoid tumours* furnished with hair; *epithelioma* and *melanotic* cancer are also found in it, and *cysticerci* have been met with. The *lachrymal gland* may be destroyed by cancer and new growths in its neighbourhood.

**Cornea.**—Although the cornea is a peculiar tissue remarkable for its richness in nerves, yet its general characters ally it closely with the fibrous textures; it is liable to a senile fatty degeneration which appears as a whitish opacity of the circumferential unused portion, generally under the upper lid; this may extend all round the eye; it is known as *arcus senilis.* The cornea undergoes several forms of *inflammation.* *Syphilitic* keratitis appears first as pale grey flecks which spread and unite to form a general subopacity like that of ground glass. *Strumous* keratitis shows pale spots, without the nodular elevations that characterise *pustular* keratitis, in which pus forms between the layers more superficially or more deeply; the little abscess bursts outwards, leaving an ulcer, or inwards, discharging the pus into the anterior chamber, where its presence constitutes *hypopyon.* *Ulcers* of the cornea arise either from such abscesses or originally by excavation of the tissue through pressure of a hair or other injury. If the ulcer is slight the clearness of the cornea may be quite restored, but if severe there remain opacities which if faint are called *nebulæ*, but if opaque and white are called *leukoma.* At the time when the ulcer extends deeply and exposes the deepest layer of the cornea this may bulge through the superficial opening, producing a *keratocele;* or the ulcer may quite pierce through, leading to discharge of the aqueous humour, when the iris is apt to fall against and unite with the cornea —*synechia;* or bulge through the hole, at the same time being covered with lymph—*staphyloma.*

**Sclerotic.**—The sclerotic, like ordinary fibrous tissue, is liable to *syphilitic and rheumatic inflammation*, the interstitial sclerotitis being the usual form in children suffering from hereditary syphilis. We also meet with *ulceration* of its surface, but much more rarely than in the cornea. In either situation the ulcer properly belongs originally to the conjunctiva.

**Lens.**—The lens is a peculiar substance, evascular, and hence not liable to active inflammatory exudations. Its changes are chiefly *passive;* its substance is liable to become opaque, and at the same time changed in consistence, constituting *cataract;* it may be *fluid*, and the colour whitish grey; or it may be *soft;* or else *gelatinous*, in this state resembling the natural consistence of the lens; or it may be *harder* than natural, and yellowish. The last form belongs generally to age, the former to the cataracts of younger people. Cataract is sometimes *congenital;* sometimes it is due to *injury.*

There is also a peculiar form of cataract which goes by the name "zonular." Some of the strata of the lens between the nucleus and the cortex become opaque, leaving the margin and central part clear. It appears to be associated with infantile convulsions and rickets.

**Chambers.**—The chambers of the eye may contain *blood* or *pus* or *hydatids.* Pus in the anterior chamber is known as hypopyon.

**Iris.**—The iris and choroid are sometimes congenitally devoid of pigment—*albinism.* The iris is liable to inflammation—*iritis.* This is *simple* or *syphilitic*, or due to extension of or sympathy with other ocular diseases. Syphilitic iritis in its earlier stages is found often to be localised on particular parts, but it is doubtful whether it has any sufficient distinctive characters. Some small syphilitic gummata have been met with in the iris. Dilatation of the pupil is called *mydriasis;* contraction of it is named *myosis.* When the iris becomes adherent to the cornea or capsule of the lens the state is called *synechia*, *anterior* or *posterior.* Little pigmentary tumours, or *melanoma*, were found on the iris, some congenital, by von Graefe; also *cysticerci* have been found in it. In short, it may have the same tumours as the choroid.

**Choroid.**—The choroid and ciliary muscle are liable to inflammation; a sub-inflammation of the choroid has been supposed to cause *glaucoma*, in which disease the eyeball becomes hard, causing pressure through tension within it, which pressure paralyses the retina. The cause of the increase of fluid which produces the tension is not so clear as its effects, which are greatly relieved by iridectomy. The choroid may be *displaced* by tumours or effusions of blood outside it. *Bone* may form on its inner surface, between it and the retina, especially about the optic nerve. We have several times seen the *miliary tubercles* described as forming in its texture in cases of general tubercular meningitis, an association very interesting through the correspondence between the choroid and pia mater. Close beneath the pigmented epithelium numerous *colloid* or *amyloid corpuscles* are sometimes found. These may *calcify;* this reminds one of the similar colloid and sandy corpuscles of the pia mater and choroid plexuses.

The choroid is the usual starting-point of *pigmented tumours* of the eyeball. Sometimes these begin in the orbit and invade the sclerotic, but they generally arise from the choroid membrane, and near the entrance of the optic nerve. They grow forward at the expense of the *vitreous* till they fill the eyeball, and burst through about the cornea. They are generally of *spindle-cell sarcoma* structure, rarely of *carcinomatous*, generally deeply pigmented; sometimes only partially so; Mr. Hulke and others have described unpigmented tumours from the choroid. The *melanotic* eye-tumours are never secondary. They are very infectious, extending along the optic nerve locally, and starting melanotic cancer in the liver and other remote parts, often very generally. These, together with the gliomas, are the commonest tumours of the eye.

**Retina.**—The retina may be *displaced* by injuries or by blood or exudative fluid or tumours between it and the choroid or sclerotic. The changes it undergoes when *in situ* may be admirably well traced with the ophthalmoscope. It is liable to *inflammation*, which may be of several degrees of severity and varieties of effect. Thus the retina may *suppurate* through injury, or large patches of it may be *grey and swollen* from similar causes; or in milder inflammations it may appear *hazy* and greyish or white; while in some such examples *hæmorrhage* occurs in its turbid tissue, or yellowish or rust-coloured *spots* appear in it around the yellow spot and optic disc. These milder forms occur in syphilis, diabetes, or albuminuria. The hæmorrhagic and spotted forms more frequently in granular kidney. The arteria centralis retinæ may suffer *embolism*, when the arterial vessels are found empty and the veins fuller than natural. Retinal *apoplexy*, or effusion of blood into the retina, is caused through obstruction to the passage of blood by pressure within or tumours outside the eyeball, or it happens in inflammation, as we have already said.

*Tumours* sometimes arise from the retina; of these some are more slow in their formation and approximate much to mere inflammatory thickenings. Other slow local thickenings become caseous in parts, and have been described as *scrofulous* deposits; but Virchow regards these as well as a large part of the more rapidly growing brain-like tumours of the retina as *glioma*. He says they arise from the granular layer of the retina, which he agrees with Max Schulze in regarding as, for the most part, of the same nature as the neuroglia or connective substance of the brain. Some of the rapidly growing tumours approach *sarcoma* in their characters—that is, the cells are no longer small, with small nuclei, as in the granular layer of the retina; but they are larger than mucous corpuscles, and their nuclei enlarge, and become multiple.

The tumours of the eye must be distinguished from tumours of the orbit; the latter arising generally from the periosteum and being bony, cartilaginous, or cancerous. There is also an orbital aneurysm spoken of.

## EAR

*Malformations.*—Supernumerary auricles have been described by Sir James Paget and others as arising in connection with the branchial clefts. They may be found in different spots on the neck. They are mostly loose pieces of integument, as our specimen shows.

**Auricle.—Hæmatoma Auris.**—The auricle suffers diseases the same as those of skin and subcutaneous tissue generally. But great curiosity and interest attach to the inflammation of the auricular cartilages and perichondrium, with more or less extensive effusion of blood, which is not infrequent in the insane, and is called *Hæmatoma auris.* There is little reason to doubt that this arises from injury; thus, cases have occurred in football scrimmages, and it is said that the change in question is perceivable on the ears in some ancient sculptures representing Roman athletes who used the cæstus. In this affection the perichondrium is red with congestion, and the cartilage soft, while in places, between the two, is a quantity of thin venous blood, which may be let out. After this the ear slowly heals, shrivelling at the same time. In old cases bony deposits have been found.

The perichondrium and subcutaneous tissue of the auricle often show a deposit of urate of soda in gouty people. This is worthy of notice, for it may be useful in diagnosis; such deposits may occur without pain. We have specimens showing tumours growing in the lobes of the ears from the irritation of earrings.

**Meatus.**—The meatus likewise suffers the ordinary diseases of the skin and subcutaneous tissue. It is liable to the occurrence of small circumscribed *abscesses*, which may implicate the periosteum, and produce ulceration and necrosis of the bone beneath. *Polypoid fibrous growths* and *sebaceous cysts* also occur there. The ceruminous glands in the deeper part of the meatus are apt to secrete an excess of ill-conditioned *cerumen* blocking up the passage.

The tympanic membrane is liable to inflammation and ulceration, leading perhaps to perforation. From its surface polypoid growths are apt to sprout; they are of the nature of large flabby vegetations.

The middle ear has a mucous lining continuous through the Eustachian tube with that of the nasal pharynx, and the inflammations it undergoes are those usual in mucous membranes. These are described as dry and moist cartarrh, and thickening of the mucous membrane itself. Little is known of these states anatomically; they are mostly inferred from the behaviour of the Eustachian tube during catheterism. Catarrh of the middle ear is supposed to be the cause of the deafness that often accompanies continued fever. The inflammation sometimes reaches the bone and causes caries or ulceration of it, with production of very painful and sensitive spongy granulations, which may present themselves at the bottom of the meatus when the tympanic membrane is perforated; these are of very bad import. By a more chronic action the ossicles are liable to be thickened or ankylosed either by bone or fibrous tissue. The incus may be ankylosed to the fenestra ovalis.

In the *internal ear* few changes have been noted. The otoconia or ear powder in the labyrinth has been found in excess by Mr. Toynbee. Acute inflammation, extending from the middle ear to the labyrinth, leads to acute necrosis of the bone around, which may extend to the dura mater or its sinuses, producing acute meningitis or abscess of the brain. A more chronic form of inflammation of the labyrinth is admitted, but its anatomical results are undecided. Thus, the changes as yet recognised in the ear are few and coarse compared with those recognised in the eye.

The *auditory nerve* has an evil precedence among the nerves of the higher senses in its liability to neuromatous swellings. Many of these, in the form of fibrous, mucous, and once or twice fatty tumours, have been recorded as causes of deafness and other symptoms.

## NOSE

Here also we find the cutaneous covering and mucous lining of the organ liable to the ordinary affections of skin and mucous membrane. In glanders pustules form within the nostrils; they break and leave ulcers in the swollen membrane. We must notice in the nose a tendency to obstinate ulceration of the mucous membrane in weakly people; this occurs in scrofulous persons, and is called *ozœna.* In some of these cases the secretion forms concrete masses, which the patients bring for inspection. The bone may become affected; but generally, when this is the case, the cause is syphilis, which is, anatomically, very difficult to distinguish from scrofulous disease. Syphilitic ulcers are found in the septum, or on the inner surfaces of the alæ nasi, in the form of roundish fungous ulcers, with yellowish crusts. If these are neglected, the bone or cartilage is attacked, and comes away in smaller or larger fragments. There is danger of great disfigurement through falling in of the septum, or destruction of the cartilages of the alæ. The septum is often associated with the changes resulting from chronic catarrh and ozæna, and perforation of it occurs in Chromate workers. We also meet with *adenoid vegetations* in the posterior nares in connection with the same disease in the pharynx of which we shall hereafter speak. *Polypi* of the nose, called mucous polypi, are frequently removed by the surgeon; they are soft, gelatinous-looking, and consist of a delicate fibro-cellular structure, composed of long filaments connected with stellate cells, forming a lax meshwork, with a thin mucous fluid soaking it. They are highly vascular, and the surface is covered with ciliated epithelium; so that it forms a very beautiful object for the microscope; if a piece is snipped off and placed beneath the microscope, nucleated columnar epithelial cells will be seen with their edge furnished with cilia in constant vibratile action; it must be placed in a little of its own juice, for if put in water the motion will stop. The growths from the mucous surface are not all of this innocent nature; we may meet with polypi which are cancerous. These are generally *sarcomata* attached by a broad base, and are connected with the periosteum or bone.

The sinuses in communication with the nasal passages sometimes have their orifices obstructed, while they become the seat of inflammatory mucous flux, so that the cavity may be distended and cause great suffering. In some cases the contents occasionally burst through the obstruction, producing periodical discharges with temporary relief.

Sometimes the retained secretion bulges the frontal sinuses either forwards chiefly, or towards the orbit. In considering such cases we must remember the small congenital encephalocele at the root of the nose. Sometimes round-worms or insect larvæ find their way into these sinuses (*e. g.* the maggots of a species of fly in South America, *Lucilia hominivorax*).

# DISEASES OF THE SKIN

We shall consider these as corresponding to the several components of the skin as far as this is practicable, dividing them into diseases of the epidermis, corium, and appendages.

## EPIDERMIS

**Atrophy.**—An atrophic state of the epidermis, together with an imperfect formation of its elements, imparts to the skin a disagreeable thin dry appearance, while the surface is chaffy, or brawny, or greasy and lustrous. We see this state most evidently in old age, or in syphilitic subjects, especially infants, and to a less degree in hepatic and other cachexiæ, in all of which states the epidermic appendages—hair and nails—suffer likewise in their nutrition, becoming thin, falling off, or losing their colour. For a description of congenital ichthyosis, or harlequin fœtus, see "Fœtus."

**Hypertrophy.**—The epidermis, like all parts composed of cells, quickly increases from over-activity in its vascular supply, so that a degree of thickening of it forms a part of a great number of diseases. Thus during the superficial bright-red blush of scarlatina new cuticle is formed, which throws off the old in subsequent desquamation. The deeper seated congestions of larger vessels in measles, &c., usually fail to do so; so also often during and after intense sweating in rheumatic, scarlet, or typhus fevers, excess of cuticle is found to displace the old, inducing partial desquamation.

But, besides these desquamations revealing the results of active congestion of the corium, thickening of the epithelium is found to constitute the anatomy of many forms of cutaneous disease. These may be diffused over a large part of the cutaneous surface, arising from a general cause, or involve only a small part, from the cause being local.

The diffused kinds are *psoriasis*, *lichen*, and *pityriasis* (an improved nomenclature making lepra apply, as in former times, to leprosy only). The papules of lichen consist of epidermic cells heaped up in and around the hair-sacs and mixed with sebaceous matter; such papules may be detached without bleeding, but when the lichenous affection is severe it may inflame the corium, and throw the whole epidermis into a morbid state, while the hair-sacs are especially diseased, and the roots of the hairs deformed. This more severe *lichen* constitutes a rare and dangerous disease, called *Lichen ruber* by Hebra and *Lichen planus* by Erasmus Wilson, and now named *Lichen ruber planus*, and *Lichen ruber acuminatus*, according as the papules are flat or rounded. *Psoriasis* is a morbid condition showing itself in excessive formations of cuticle, whereby patches of scales are produced; and, according to the amount and shape of the patches of disease so produced, various trivial epithets are given to the affection. But in our post-mortem examinations of such patches we have, like other observers, not been able to find any morbid appearances, except the scales themselves, composed of compacted epidermis. The red patches of the cutis, on which they are borne during life, become pale after death. As to pityriasis, we shall here only allude to that formidable disease *Pityriasis rubra*, described by Hebra, in which a general redness of the skin, with a universal exfoliation of the epidermis, make the only anatomical sign of a disease that is almost uniformly fatal. We have models of the disease under the name of *dermatitis universalis.*

When the epidermis is congenitally thick and rough, the name *ichthyosis*, or fish skin, is used; some remarkable examples of this may be seen in our wax models and drawings and also in a piece of skin removed from a patient so affected. The cuticle projects in little papillose processes, which, lying side by side, produce an appearance like the pile of velvet; or, becoming thicker in some places, grow so large that they resemble warts. In less degrees there is merely a thickened scaly state of the epithelium.

There is a localised hardening of the skin, sometimes met with in children, and called by Kaposi *Xerodermia.* Of *local* hypertrophy of the cuticle we see examples in *callosities* and *corns* arising from pressure. The former are met with on the palms of the hands of those who work hard, as watermen, &c., thick layers of epidermis being thus produced. Corns are mostly seen on the toes, and result from tight or ill-fitting boots, but they may form on any projecting parts; they consist of accumulated masses of epithelial scales, which lie superimposed, and rise up one over the other until they form a column, whose lower part sinks down upon and into the true skin. We also see remarkable *horny growths*, composed of a collection of epithelial scales; these do not rise, as far as we know, from pressure, but from some other local irritation; a vast production of diseased epithelium takes place, and rises up by continued additions from beneath, until, in time, long spiral projections are produced, resembling horns, or like the chemical toys seen under the name of Pharaoh's serpents, which grow more rapidly, but in a very similar manner. If examined, they are found to be composed of epidermic scales, massed together in layers; there is also amongst them a little fatty or sebaceous matter, but we do not know that they always have their origin in sebaceous follicles. There is a specimen in our Museum of a horny growth that came from an old woman, who, about twenty years before, noticed some scabs on her head and body, and these gradually increased in elevation during some years, until three horns appeared. These spontaneously dropped; but they all began to grow again, and then a second time fell off; and ours is one of the last crop. A smaller one Mr. Cock removed from a man's lip. These must not be confounded with very similar looking horny warts of epithelial cancer.

**Alterations in Colour.**—These alterations in the skin chiefly depend upon changes in the epidermis, though sometimes a deposit of pigment occurs in the corium; and the colour of the skin is influenced also by the state of vascular supply in the corium, by effusion of blood or petechiæ, by fatty degeneration, &c. All these simple alterations of colour go by the general name *maculæ.* It is in the layer of young cuticle, called the *rete mucosum,* that the colour lies which gives the peculiarity to the dark races of man, but some pigment is always found in the papillary layer of the corium. If one examines our specimens of the skin of the negro, or makes sections of them, there will be seen, immediately beneath the epidermis, and corresponding to the papillæ of the skin, a dark layer of cells.

In various morbid conditions of the skin a pigmentary deposit takes place in the same position; but the most remarkable is that known as *melasma suprarenale,* or Addison's disease, where sometimes the whole integument becomes coloured, so as to resemble exactly that which belongs naturally to dark races, closely resembling the colour of a creole or quadroon; indeed, in some cases no difference could be observed between persons so affected and those born with an admixture of dark blood. If there should be any doubt upon the matter, such a case can be clearly distinguished from one of mere superficial staining of the skin by making a section, and observing whether the dark layer is under the superficial epidermis, seated close down upon the true cutis, and running everywhere in the course of the papillæ. In microscopic sections the peculiar colour can be seen. Although the name melasma has been given by some, the term *bronzing* (cutis ænea) has been adopted by others, as the colour is of a greenish-brown hue. The colour is universally diffused, and is not in patches; but the bends of the joints, the scrotum, the areolæ of the breasts, &c., grow much darker than other parts. The depth of tint may vary a little from day to day, apparently through variations in the vascular supply. The dark colour persists after death. This colour is often mistaken for jaundice, though it is very unlike the common yellow hue of jaundice. But in very long-standing obstructive jaundice the skin wears a very dusky hue; it is, indeed, pigmented with black pigment in much the same way as in Addison's disease. This blackish change in jaundice deserves more attention than it has yet received.

In pregnancy a similar darkening occurs in some localities, as the face, areolæ, and linea alba; also in cases of enlargement of the glands and other abdominal diseases. This blackening is called Nigrities, so is a similar pigmented state found on the scrotum. The name *chloasma* is given to patches of a size from that of a crown piece to the palm of the hand, or larger, on the face or trunk, or especially on the extremities; these have been found connected with chronic hepatic disease, syphilis, and other disorders.

The name *Ephelis* is applied especially to a temporary discoloration of the skin from exposure to the sun, as the name implies; while *lentigo* is used for permanent *freckles.* The term *pityriasis* has been used rather loosely to denote a desquamation of the superficial epithelium. *Pityriasis versicolor*, the variety which is distinctly recognised, is due to a vegetable growth.

There is another remarkable staining of the skin, which is called *Ephelis ignealis*, or *ab igne*, and which is represented in our models. It appears to occur in invalids who have sat for a long time before the fire and scorched their legs and arms; especially the former. A number of reddish or brown lines are seen coursing along the front of the leg, in the position of the superficial veins; the fact of lines exactly resembling these occurring as a post-mortem appearance, due to an imbibition of the colouring matter of the blood in the surrounding texture, would make it appear as if some similar cause was in operation in these invalided persons—that, in fact, some extravasation occurred from the vessels through the heat, so that a permanent discoloration took place in their course.

*Argyria.*—Discoloration arising from the internal use of *nitrate of silver* is very remarkable, and when once seen can never be forgotten, as the black-lead-like half-metallic lustre of the skin, and especially the face, is very striking. In these cases the internal organs have also been found discoloured, *e. g.* the mucous and serous surface of the alimentary canal and parenchymatous organs.

*Albinismus* is the state where colour is congenitally altogether wanting in the skin, as well as in the hair, eyes, &c. *Vitiligo* is the term formerly employed for an acquired and partial want of colour, but the word *leucoderma* is now more generally used. Such conditions sometimes are so extensively diffused as to make the individual look "piebald." It is often associated with a deepening of the colour in the intervals, making it difficult to say whether the whole appearance is due to local excess or deficiency of the pigment; other conditions we shall mention when we speak of leprosy.

**Parasitic Growths.**—*Pityriasis*, or *tinea*, *versicolor* is due to a parasitic fungus known as the *Microsporon furfurans.* The disease is a very common one, and easily recognised in those buff-coloured patches so frequently seen covering the chests of the poorer people, especially of those who constantly wear flannel next the skin, and do not indulge in habits of cleanliness. If some of these yellow parts of the skin be scraped on to a glass slide, and the latter be placed beneath a microscope, the epithelial scales will be seen covered with the sporules of the fungus. In order to discover them a drop of water should be placed on the specimen and then a little liquor potassæ, or carbonate of soda, in order to render the scales transparent, when the sporules are readily seen. These are small round globules adherent to the scales, and besides them will be seen some long cells placed end to end, which form filaments, or the *mycelium*, as it is called.

## CORIUM, OR TRUE SKIN

The anatomical condition of the skin is generally little considered in distinguishing and treating its diseases; and this, no doubt, is very natural when it is remembered that the cutaneous affection is often only a part, and sometimes a small part, of a severe constitutional disease, as, for example in the exanthemata. There are many affections of the skin, however, which appear to be strictly local, though, as to many other so-called skin diseases, it is difficult to say whether the skin alone is at fault, or whether the eruption is not rather a mere manifestation of some inward and more hidden malady. Whatever may be the cause, however, of a skin affection, it would be our place to show the anatomy of the disease, apart from any connection it might have with an internal disorder. Having said this, it might be expected that we should demonstrate what the peculiarities are, but this, unfortunately, we are unable at present to do in all cases, although we have no doubt the real change in the skin, in all cutaneous affections, will at some future time be discovered. We can see how, in the exanthemata, a mere difference in the state of congestion (due, of course, to some anterior condition) may cause a variety in the character of the eruption—as, for example, in *scarlatina* and *measles*. In the former, as we have before said, the congestion implicates the minute capillaries of the surface; in the latter it affects deeper-seated veins, whose fields appear as distinct dull blotches; and in this way, no doubt, a certain fixed anatomical condition exists in scarlatina, another in measles, another in *roseola*, &c. In *erythema* the redness is still more even. In all these rashes the blood is still held within the vessels of the skin; but if the blood be diseased, and the due relation be lost between the vessels and the tissues, then it bursts forth, and produces small effusions in the superficial layers of the cutis, known as *petechiæ*. Scarlatina, measles, smallpox in its first stage, may assume this character, but it is in typhus fever that it is more commonly seen. In this disease the dark-coloured spots of the *mulberry rash* (as it is called), after a few days, cease to fade on pressure, and towards its termination, especially if it be fatal, the blood actually transudes from these congested spots, and petechiæ are produced. In the rash of enteric fever the rose-spots never go beyond the state of congestion; the blood being still in them, so that it is driven out of them by pressure. If blood transudes without any known cause, the name *purpura* is given to the disease.

We have considered the rashes as inflammatory irritations with only the anatomical results of active fluxion in their simpler and milder forms. But sometimes exudations do take place, and then we can call them none other than inflammatory. Thus, in scarlatina, raised papules on the skin are often observed, and occasionally in these a small quantity of fluid is formed; and probably in all such acute papular eruptions, even if no fluid is seen by the naked eye, some exudation really is present.

In fluxion to the skin, however, which constitutes a rash, there is usually simply a hyperæmic condition of the vessels; but in another class of cases, where the skin itself appears to be the seat of primary irritation, often from external causes, we find a more severe action in the corium, producing a papular eruption, being at least one form of what was called lichen. This has been called by some eczema, and the term lichen limited, according to Hebra's plan, to the more chronic papular epidermic heaps already described. This form of apparently solid little elevation in acute cutaneous irritation will then take its proper place, as belonging to the early stage of *eczema*.

The next stage of eczema beyond this is shown in an actual production of serum. We may watch such a process on ourselves; for example, when the hand is exposed on a hot summer's day to the sun's rays, we may first perceive a redness on its surface, and this is accompanied by heat and pain; we may afterwards perceive a lichen-like papular eruption (in the East called *lichen tropicus*, or prickly heat), at a later stage small vesicles of eczema appear. These vesicles are small collections of fluid under the scarf skin, lifting it; after a time they will burst the cuticle and discharge their contents. Such eczema appears to be the simplest inflammation of the skin, through irritations to its surface. We see modified forms of it in *grocer's itch* from the action of sugar, in *baker's itch* from flour, also in dirty heads, &c., the parts affected being those annoyed by the foreign irritant. Of course, it will be recollected it is only some of these kinds of people suffer, so that the sufferers must have a peculiarity of skin predisposing to the annoyance, which peculiarity may be inherited, and thus a disease plainly due to external irritation may run in families.

A totally different kind of inflammation of the cutis produces vesicles as in *Herpes*; it arises from causes differing from those of eczema. The vesicles are large, but are similarly elevations of the cuticle; some ulceration of the cutis may follow them if they be severe. These causes are interesting, because of the frequent occurrence of herpes of the nose and lip in pneumonia, in cerebro-spinal fever, in the malarious fevers of America, &c. The herpes forms a very curious sign of relation between these, especially when it is seen how very frequent this herpes is in some people, and how identical it is anatomically with *zoster*, which differs only in following the distribution of a nerve, an irritation of which is often obviously the cause of the eruption. Another curious example of such vesicular eruption is seen in the condition called *hydroa*, which attacks the forearm and face symmetrically in the form of eruptions of vesicles, limited often most exactly to the distribution of the fifth and other nerves. We have seen the vesicles become solid papilliform nodules, which bled when pricked. They afterwards withered away. Some think this hydroa is only a product of iodide of potassium. We doubt this view, having seen it arise when no iodide was being used. Iodide and bromide of potassium do, however, produce a pustular eruption. A yet different cause reveals itself in the production of the large bullæ of *pemphigus* or *pompholyx.* In all these vesicles the fluid is generally alkaline.

Now, as in other parts, a different quality or greater intensity of irritation will produce *purulent* matter in the vesicles, and thus a pustule be formed, as in *ecthyma*, *impetigo*, &c. Though it is interesting to consider as far as possible what are the exact anatomical conditions of the skin in its various diseases, yet this, of course, still leaves unexplained why a particular amount of inflammation should occur in one case and not in another, or why the rash is disposed to assume one form in one case and another in another, &c. These differences make evident unknown differences of cause.

In *vaccinia* and *variola*, as we know, the vesicle is depressed in the centre and divided into a number of cells containing a specific fluid. It begins as a papule and becomes a complete pustule in eight days. It is characterised by being flat and depressed in the centre, or, as it is styled, *umbilicated.* The cutis vera pours out a semi-solid lymph, which being held down in the centre, a number of folds or loculi are formed in which the more fluid exudation is contained, so that both the variolous and vaccine pustule must be punctured in many places to evacute the chambers. The depression in the centre corresponds usually with the position of the hair and sebaceous follicles, but the same formation is seen where no hair exists, so that the shape of the pustule must depend rather upon its situation over certain groups of papillæ and ducts, and these determine the presence of the stigma which holds the centre down. Some have said that the mere shrinking of the cells will account for the depression. The form of the pustule is seen in the characteristic vaccine cicatrix which follows: this is round, radiated, and foveated or dotted.

When the true skin and subcutaneous tissue are inflamed throughout, *phlegmonous erysipelas* is produced. In superficial erysipelas there is merely a swelling and slight exudation in the form of vesicles or bladders beneath the cuticle; but in the phlegmonous form, the whole depth of the skin and subcutaneous cellular tissue may become involved, so that purulent matter is formed in the deep parts, and much of the texture may die and require to be discharged in the form of a slough. A local form of a like inflammation is seen in *furuncles* and *carbuncle.* It must not be thought that these always commence in the deeper parts, for we often observe that they begin on the surface and spread inwards; the sufferer first perceives a redness and feels an itching, and then presently a small vesicle forms, the who being superficial; but in the course of a few hours, the swelling and induration has increased, until he discovers that the skin is considerably involved, when suppuration occurs, perhaps, with the discharge of a slough.

The distinction between boil and carbuncle is usually drawn by the number of openings formed, single in a boil, multiple in a carbuncle. Hebra, we think, improves on this distinction by making the presence of a slough the sign of carbuncle, while the boil or furunculus only suppurates without sloughing. Either means of distinction is artificial, for the two conditions graduate into each other indefinitely. A large carbuncle brings with it danger of exhaustion or pyæmia. Boils are generally rather troublesome by their repetition. This may be due to some external irritation as when they arise in and complicate eczema, scabies, prurigo, phthiriasis, &c., or else it may be founded in a special disorder, of which it is the sign, when it is called *furunculosis.*

We must draw attention to the terrible form of boil on the face, generally seated on the lips, sometimes called *malignant carbuncle* or *charbon;* the facial veins become implicated in this disease, and suppuration extends along these to the cranial sinuses, causing death with cerebral symptoms sometimes rather suddenly.

Probably this and some other kinds are due to the absorption of animal poisons; as, for example, the *charbon* just mentioned, which is thought to arise from infectious matter introduced through the bite of a fly, or the *malignant pustule* of former days affecting generally the hand or upper extremity. Probably some of these diseases were the same as that which is now especially called *anthrax*, and arising evidently from the poison of the *cattle disease* known as *milzbrand.* It is a form of disease with which the surgeons of Guy's Hospital are quite familiar as occurring amongst the tanners of Bermondsey, who handle foreign and diseased hides.

If the skin be wounded and poisoned, a vesicle forms, soon to be followed by œdema, with inflammation of the skin and subcutaneous tissue, until a large carbuncle is formed. It is characterised by being a hard swelling of a purple colour with a depressed eschar in the middle and surrounded by vesicles. The parts are indurated, œdematous, and inclined to slough; it therefore can in no sense be called a pustule. Dr. Charlewood Turner made sections of the anthrax, and found the Malpighian layer of the skin raised by a serous exudation, crowded with leucocytes, and full of characteristic bacilli, the large *Bacillus anthracis.* These were aggregated in masses amongst the sheaths of the hair-follicles and did not affect the deeper parts of the skin. If these boils be excised, the patient generally rapidly recovers, although, strange to say, swarms of bacilli may still remain in his blood. This anthrax appears to be a local manifestation of the fatal disorder known at Bradford as the *woolsorters' disease.* The men employed there in sifting the newly imported wool inhale the deleterious matter through the lungs, and quickly die of blood-poisoning. The interior of the body of those who die of this disease shows peculiar changes in the stomach and intestines, to be hereafter mentioned—a black sloughing form of anthrax on the mucous membrane.

*Glanders* is produced by inoculation or contagion from horses affected with the complaint. When the system is poisoned, vesicles form on the skin somewhat resembling those of varicella; then pustules or bullæ come, which in appearance are not much unlike those of variola. Besides these there are usually swellings or lumps in the subcutaneous tissue. These are well displayed in the Museum models. Occasionally, instead of the disease being quickly fatal, it is prolonged over a long period by the presence of chronic sores. For further particulars see "General Diseases."

Amongst the chronic effects of animal irritants are the wart-like swellings so common amongst those who are engaged in post-mortem examinations. We have models of the affection, and named it *Verruca necrogenica.* It is also known as the anatomical wart. There is no reason to believe that it contains the tubercle bacillus, as has been suggested.

**Morbid Growths.**—Several of the diseases which we have placed under this head might, we think, with almost equal propriety, have been regarded as chronic inflammations; but the same difficulty is found in the skin, as elsewhere, in defining what is an inflammatory product and what is to be considered as a tumour. For instance, we have just now spoken of phlegmonous inflammation of the skin and cellular tissue. But several chronic affections which we are about to consider might be looked upon as a slower process of a somewhat similar kind; indeed, the results of inflammation are so much like tumours that they often cannot be distinguished.

For instance, we have a drawing of an arm, showing a cicatrix following phlegmonous erysipelas, but from the omission of an explanation of it, it had been considered as a case of *keloid*, a disease which is eminently chronic, but which, in the course of time, produces the same wasting of the cutaneous tissues and cicatriform appearance. In another form of disease, which we used to call the *keloid of Addison*, and which is represented in several models and drawings, there appears to be a chronic formation of lymph or inflammatory matter in the skin and subcutaneous tissue, so that a swollen or brawny appearance is produced.

This disease is now included in the more general name *scleroderma;* an exhaustive paper on which cutaneous affection will be found in the 'Guy's Hospital Reports' by Dr. Fagge. It produces a rigid state of the parts without fever or other signs of active inflammation. It generally spreads from the spots first attacked, and is more common on the thorax and upper extremities. The deeper layers of the skin become hard, whilst the epidermis is smooth so that the skin cannot be pinched up in folds. It is sometimes well defined; in other cases the disease passes gradually into the healthy skin. In the course of time a wasting or shrinking takes place, so that a cicatriform appearance is left resembling the result of an inflammatory abscess or healed burn. Whilst including the scar-like form of keloid described by Addison under this general term, it is advantageous to preserve this latter expression for the more isolated rounded tumour described by *Alibert*. These project from the skin, are of a bright red colour and shining; they are vascular, and when removed are found to consist of a dense fibrous structure springing from the skin. These tumours often send off claw-like processes, and then, combination taking place around them, they approach, in appearance, the other variety mentioned. They have been described as primary and secondary, the latter term being referable to those cases which spring up in the site of a cicatrix of an eruption or other affection of the skin. Some authors have never met with keloid under any other circumstances, and would therefore believe that such growths are always secondary. Cases, however, have been been described where the growths have apparently been primary, and sprung up in the healthy skin. One of the most remarkable and interesting facts concerning keloid and some forms of scleroderma is that the disease will spontaneously undergo involution, and altogether disappear. The thickening of the tissue may pass away, and the atrophied portions of skin again fill up, until an almost healthy condition of the skin may again appear, or at most the only trace of the disease discernible be a few simple scars. Dr. Fagge described cases undergoing cure, and Dr. Goodhart has watched, for several years, a case of keloid where large masses of adventitious tissue have disappeared, until nothing but scars have at last remained.

*Morphœa.*—This name is given to white shiny patches on the skin, which Dr. Fagge believed were of the same nature as scleroderma and not lepra, under which name they had been generally included.

We might mention in this connection the *striœ atrophicœ* met with occasionally on the limbs and other parts and having a close resemblance to the *lineœ gravidarum.*

*Leprosy or elephantiasis.*—Unfortunately under this name two different maladies have been described which are pathologically quite unconnected with each other, although both occur in hot climates, and very often in the same locality, as in the West Indies.

The one is a *local* affection, the other *constitutional;* in the one the part affected, usually the leg, swells to an enormous size during a period of several years, while the patient is in good health; this is the "Barbadoes leg," originally called *elephantiasis Arabum*, and called in our Museum *elephas.* If we look at the specimen in the Museum we shall see that its right to a title founded on likeness to an elephant's foot is beyond dispute. The disease is due to a simple increase of the skin and subcutaneous tissue, whereby the former is hypertrophied and an immense thickening is produced beneath it, and, at the same time, the fat is often much increased in quantity, although it is sometimes much diminished, being compressed by the new-formed fibrous tissue. The skin also becomes brown, as seen in our models, and a number of fissures occur, which subsequently enlarge to oozing ulcers. This disease of the leg, as we have said, attacks those living in hot climates, especially the lower orders of the people, and mainly those of uncleanly habits. It commences as an œdema, and is then curable with care. It is probably the same disease as affects the genital organs in India, producing elephantiasis scroti. Although the disposition to it becomes excessive in the tropics, we are by no means free from the disease in this country, both as to the legs and genital organs. During the last two years we have had three cases of this affection in the hospital; the disease was of many years' duration, and the general health of its subjects did not suffer. In two of these cases after amputation (the specimens may be seen on the shelves, as well as another of the true West Indian variety), we had an opportunity of making a section and observing the structure; the cuticle was very much thickened and of a dark colour, the cutis was also thickened, and beneath there was a quantity of adventitious fibrous material and fat, but no elements different in kind from those normally present. The bone was healthy, and the veins leading to the diseased parts were exceedingly thickened. The state of the lymphatics was not made out.

In this form of elephantiasis, as seen in India especially, if it attack the scrotum, the lymphatics are often enlarged and a constant drainage of lymph may take place from them. In this are found multitudes of the *Filaria sanguinis hominis.* Mr. Croft had a case where the thigh was affected with this disease, and it discharged enormous quantities of milky fluid.

The other disease, which was called *elephantiasis*, was distinguished as *elephantiasis Græcorum,—leprosy.* It occurs in the tropics; but it is not limited to hot climates, being a terrible scourge in some districts chiefly maritime, in Norway; indeed, the fullest account we have of it is from the celebrated work of Daniellsen and Boeck, which embodies the result of an inquiry ordered by the Swedish Government. To avoid confusion it is much better to confine the term elephantiasis to the local affection just described, and to name the disease under present consideration by the term *lepra.* This term is, indeed, now very generally so used, and its old employment for a variety of psoriasis is done away. The prevailing opinion is that this lepra is allied to, if it be not the same disease, as that with which the ancient Jews were afflicted, though Hebra gives curious reasons for the opinion that this was often scabies. True leprosy, as met with in the East and West Indies and in Norway, &c., appears under two forms, though these are not so separate but that they are found on the same individual. In the first form protuberances appear on the face and elsewhere. This kind is popularly called leprosy, or in scientific nomenclature *lepra tuberculosa;* the other form is attended not so much with swelling as with stiffness of the limbs and subsequent wasting and falling away of the digits; it is known familiarly as the *joint evil*, but, owing to the numbness which attends the disease, it is scientifically called *lepra anæsthetica.*

The first form, or tubercular leprosy, is occasionally seen in the hospital. The face and hands are chiefly affected. The anatomy of the disease shows the presence of a large quantity of a new formation in the tissues of the skin, extending from the surface of the corium down into the subcutaneous texture. The new matter is composed of cells of round or spindle shape which when seen in crowds look like pus-cells, but in the spreading margin are larger and caudate.

Dr. Thin, who has paid much attention to the subject, says that when a section of leprous skin is made and stained with fuchsin, the true leprous cells stand out; the smaller ones resembling white blood-corpuscles. The dye leaves unstained the surrounding connective tissue. The cell is coloured, but not the nucleus, and in the cells bacilli may be seen. In the connective tissue and other parts of the skin they are not observed. In appearance the bacilli resemble somewhat those of tubercle. If the skin be not ulcerated, they are not found; the epidermis contains none, and therefore appears to form a covering impermeable to the special parasite of leprosy. In some cases the bacilli have been found in the blood.

Virchow placed this leprous tissue in the class of granulomata or growths resembling granulation tissue, along with syphilitic tubercles and lupus, which superficially so much resemble leprosy. The new matter forms knots or tubercles in the skin, the cuticle over these knots becomes shiny and thin, and the glands and hair perish. But the disease rarely invades the scalp; it is only the brows and cheeks that become bare. When thus affected, the skin becomes swollen and indurated, while protuberances form on it, so that the most frightful and loathsome appearance is produced; the face expanding and the features becoming bloated, with a grim and sinister aspect, better deserving the name *leontiasis*, which is now given to it, than the old term elephantiasis; the lips become much enlarged, the alæ of the nose expand, the eyelids thicken, and large folds of skin hang down on the cheeks and forehead; the hands and feet are generally similarly affected. The disease is not, unfortunately, altogether unknown as arising in our own country; but some believe that all such cases are only forms of syphilis. A few years ago, however, a very interesting case of true leprosy occurred in a man who had never been out of London; the models and drawings of his face are in the Museum. It was affected in the manner we describe, producing this frightful disfigurement. He died with an ulceration of the larynx, a complication which is often the immediate cause of death in Eastern climates. Very often, the skin of the whole body, although not thickened, is discoloured, and covered by white and brown spots. The word *morphœa*, as we just now remarked, is used in our Museum for circumscribed scar-like patches, generally on the forehead, which patches, Dr. Fagge thinks, are connected with scleriasis rather than leprosy, but many still consider it a form of this disease.

The other form, or *lepra anæsthetica*, is characterised by the shrivelling away of the fingers and toes, as seen in our models. In the first instance a mere numbness or loss of feeling indicates the onset of the disease. This condition is shown to be caused by a cellular material formed in the trunks of the nerves. We have already mentioned the observations of Virchow and Carter on this subject under "Nerves." The skin of the limbs, especially of their ends, is in this variety occupied by the morbid product more uniformly; it contracts upon the textures beneath and ulcerates, so that the bones are reached, and phalanx after phalanx dies and drops off, and thus the fingers and toes shrink away, leaving nothing but stumps, which mutilation may reach to the loss of the forearms, the general health of the sufferers, meantime, remaining tolerably good. For further particulars of this interesting disease the work of Danielssen and Boeck may be referred to.

We cannot dismiss this subject without drawing attention to some other obscure allied affections, which appear either as occasional diseases in our own or other countries, or which are found in limited localities under special names. Virchow's great work on 'Tumours' contains a most interesting review of these disorders, which he groups together as leproid, lupoid, or syphiloid, in his class of tumours having the structure of *granulation tissue.* The principal of these is *Radesyge,* occurring in Norway; an affection generally of the extremities, in the integuments of which soft tubercles form and ulcerate quickly, differing from leprosy in being limited in area; the tubercles being larger, and many deeper seated; they also ulcerate more swiftly. Hebra can see nothing in these but sometimes syphilis, and sometimes serpiginous lupus; and Virchow has, we believe, come now to the same conclusion. *Yaws* or *Frambœsia* appear as growths resembling condylomata in structure and position, being red raspberry-like warts, which ulcerate and lose their skin; they may group themselves into larger masses, called "mama yaws;" they are contagious, and heal sometimes spontaneously. The disease belongs to the tropics in the Old and New World. There is the same question as to their syphilitic or leprous nature as in the case of Radesyge. Sydenham set them down as syphilitic, and there are many who would do so now. The *Sibbins* or *Sevins* of some districts in Scotland is more generally regarded as syphilitic; it is very like both yaws and Radesyge.

Besides these better known diseases there are other cutaneous tuberous affections which are regarded commonly as peculiar local manifestations of syphilis—confidently by some, while others think this doubtful, because often no history of syphilis exists, and when it does, there are perhaps no other cutaneous results of the syphilis, and there is a long interval since the primary disease. Yet further, there are on record peculiar forms of tubercular affection of the skin which it is useful to bear in mind when any remarkable case occurs. Thus, there was recently a man under Mr. Durham's care in Lazarus Ward, on whose skin were very remarkable tubercles and tubers, some of large size. They were red and shining, and rose rather boldly in relief; when larger, these surfaces ulcerated, producing altogether a very peculiar appearance of large spongy masses, with a soft-looking, grey, ulcerous surface. Some of them healed spontaneously, others dipped deeply into the subjacent textures, causing frightful gaping sores. In the end the poor fellow died worn out, and we found the tissues of the masses to correspond exactly with the "granulation" structure of lupus and lepra. This case is evidently of the same nature as those allied to frambœsia, described by Alibert under the name of *mycosis fungoides.* Anti-syphilitic treatment had no important effect on the disease. Very similar must have been the appearance of the Irish button scurvy, which, however, was contagious, or of the Aleppo evil, Delhi boil, &c., which are ulcerating cutaneous tubers, thought to be due to drinking unwholesome water; but we have never seen these. Mr. Davies-Colley also had a case where there was an extensive ulceration of the skin, which he thought to be the result of a breaking down of sarcomatous growths.

Fibrous deposits appear in the skin and subcutaneous tissues in the form of *syphilitic tubercles*, which may sometimes, as we have seen, resemble leprosy very closely. These have the characteristics of gumma in other parts. The word tubercle is not used in the same sense as that in which we speak of tubercle in phthisis; but in the sense in which the dermatologists define tubercles, merely from their external form; thus, "solid swelling of the skin; size from a lentil to a hazelnut, and covered with epidermis." We find such tubercles also very often in *acne indurata.* Fibroid material makes up more or less of them as a new matter uniting together the inflamed and suppurating sebaceous glands.

Under the name lupus are included two affections which apparently have nothing in common—simple lupus and erythematous lupus.

In the *lupus erythematosus* the skin of the nose or face becomes red and scaly owing to a growth taking place beneath, and slowly spreading. A section of the skin shows a mass of cells with dilatation of the blood-vessels and changes in the sebaceous glands, so that the name sebaceous lupus has been proposed, or *seborrhœa congestiva*, by Hebra.

*Lupus vulgaris* is an inflammation of the skin and mucous membranes of a special kind in which granulation tissue is formed. The deep layers of the cutis are seen occupied by a small-celled infiltration extending along the course of the blood-vessels. These form nodules from which there can be expressed a fluid, to use Hutchinson's phrase, like apple jelly. The disease spreads, and as it does so, the first affected parts shrink, leaving a cicatriform appearance, which is very characteristic of the disease. If the new formation softens and disintegrates, then an ulcer is formed. The mode of formation of the granulations, and the presence of giant-cells, suggest its relation to tubercle, and this correspondence agrees with its general appearance and character, which have made the French class lupus amongst the *Scrofulides*. Dr. Thin, who has paid careful attention to the histology of lupus, describes it as a cell-infiltration penetrating the corium uniformly, beginning in the form of nodules which produce elevations to be felt by the finger. These afterwards join, and fresh nodules form at the periphery of the affected part. This is what Virchow calls granulation tissue. He found the cell formation following the course of the blood-vessels, and as to the character of these cells he thought they might be the white corpuscles, as they exactly resembled them in size. Very often the blood-vessels have become degenerated, stiff, and brittle. Giant-cells have frequently been found, containing many nuclei and having long processes; their presence, as before said, has suggested the relation of lupus to tubercle. It has been argued against this that giant-cells are not always the same, and in this instance differ in character. Dr. Thin offers a theory, which is that the adventitia may form the external processes of the cell and the endothelium of the intima its cells, and therefore the origin of lupus is in the blood-vessels. Lupus affects the skin, more especially the face and extremities; also the tongue and mucous membranes. A bacillus is sparingly found in the cells which form the new lupoid tissue.

*Rodent ulcer* has usually thin edges, and is unlike cancer or lupus in no granulations being seen. It is generally covered by a reddish-brown scab, and affects usually the face. Dr. Thin found, in some cases, a markedly thickened edge, and in this was a growth of cells putting on a columnar arrangement; there was no appearance of laminated corpuscles or overgrowth of epithelium. The connective tissue became wasted as well as the blood-vessels. In some cases the sweat-glands were affected, the duct being dilated and filled with cells. He believed, therefore, that some rodent ulcers had had their origin in disease of the sweat-glands, in others in the sheaths of the hairs. This description confirms the opinion of the best authorities, like Paget and Hutchinson, as to the non-cancerous nature of rodent ulcer. There are those, however, who have studied the subject, as Warren of Boston, and Hulke, who maintain its connection with epithelioma. They describe the edge of the ulcer as composed of cells with a cylindrical arrangement; at the same time they find clusters of cells matted together, and having altogether an epithelial type.

We might allude here to the lupoid affection of the septum of the nose to which Hebra gave the name *rhinoscleroma*.

In *molluscum fibrosum* pendulous tumours grow from the skin, sometimes in great numbers, and often as small warty tumours from the nose. They are described by Virchow as composed of tegumentary tissues, and allied to elephantiasis growing from the connective tissue in the subcutaneous region. In one case Dr. Fagge found a ramified sebaceous gland in each small tumour. Such a large gland is held to characterise *molluscum contagiosum*, and to be absent in the fibrous form, yet the large tumours in this case were fibrous. In all probability the glands were accidentally affected, for as a rule *molluscum fibrosum* and *molluscum contagiosum* are totally different diseases. We have already spoken of a state of the thigh resembling elephantiasis, in which the lymphatics are likewise enormously enlarged, and may burst, producing lymphorrhœa.

*Condylomata* are new growths of the papillary layer of the corium; they, too, consist of tissue resembling that of granulations. Two kinds are distinguished: the first, called broad condylomata, or *mucous tubercles*, are the wide, flat, and softer varieties of papillose elevations of the corium; these are generally met with near the genital organs in connection with syphilis. The other kind are the acuminated condylomata, which are not specific, but due to irritation from discharges; these are of more slender figure, and made chiefly of epithelium. *Warts* are new papillary growths of acuminated form, consisting of masses of epithelial cells growing in the form of a cone, in the midst of which is a loop of blood-vessel. Sometimes one cone sprouts out of another, and this indicates their mode of formation; for although the papillæ of the skin may, in the first place, determine the papillary form, yet afterwards processes shoot out and grow from the old ones—a fact which can be easily made out by careful examination. Although the papillary part is composed of epithelium, yet there is often a substratum of new fibrous structure, and sometimes an actual tumour, as seen in the warty growths from the labia pudendi.

We may here mention that curious condition, called by its discoverers, Addison and Gull, *vitiligoidea*, but which is now known by the name of *xanthelasma*. This has recently been shown by several cases in the hospital to be a result of long-standing jaundice from any cause. Severe itching of the skin has usually preceded it. In some cases of jaundice we have known the whole epidermis curiously dry, so that a scratch made a chalk-like mark upon it. The xanthelasma appears in two forms, *x. planum* being an opaque yellowish-white patch, not elevated. The pigment in the patch is gone and a quantity of fat-grains are present. In chronic jaundice this state affects the palmar surfaces of the hands, the eyelids, ears, scrotum, &c.; but one may meet with patches near the inner canthus on the upper eyelids, when there is no evident hepatic derangement. Mr. Hutchinson has collected many such cases; they were known to and figured by Rayer. The other form is called *x. tuberosum*, from the presence of tubercle-like knots on the knuckles, elbows, &c.; these knots are composed of fibrous tissue, with a great quantity of granular fatty matter. Xanthelasma is not limited to the skin; we have found it in the lips, the gums, and the trachea; also similar opaque patches we have seen on the spleen, and in the lining of the gall-ducts in these cases.

*Cancer* implicates the skin both primarily and secondarily. When a cancer grows in a superficial organ, as the mamma or testis, or a lymphatic gland or bone, the skin becomes in time adherent to and infiltrated with the growth, looking coarse, dark coloured, and vascular, and feeling hard. This is often one of the most important signs of the malignant nature of a growth.

The commonest examples are met with in extension of carcinoma mammæ, especially after the removal of the breast, when the disease may be seen returning in the skin in and around the cicatrix, as a number of hard nodules, which, when examined, will be found to show compact epithelioid cells embedded in the meshes of a fibrous matrix, which is principally at least composed of the natural elements of the skin itself, so that when the growth has sprouted out further away from the integument, it is softer, the matrix then being an altogether new formation.

But the proper cancer of the skin is *epithelial cancer*. The appearance of this is peculiar when seen with the naked eye; instead of showing a vascular juicy structure, like ordinary cancer, it is dry, friable, and has a curdy look. The main seat of all such growths is an epithelial surface. In the skin it generally affects the papillary layer, and then the papillæ grow out into warty forms, often very complex. The warts may be dendritic and widely spreading, or they may unite to form a horn-like sprout not to be mistaken for the simple epithelial horns we have before mentioned. In other cases, but more rarely, the cancer is in the deeper layer of the cutis, and then the papillary layer spreads with no warts or but few warts over the globose or conical mass of the cancer. When we examine any of these epithelial cancers we find, in sections of the warty layers, that the slice is composed of various conic sectional figures from the cut papillæ, with some old shed epithelium and dirt filling the occasional intervals where the papillæ were not in close contact. The papillæ are composed of a central vascular core, around which are large flattened epithelial cells with large polynucleolated nuclei, and with edges toothed to fit each other. The central core is also, except the vessel, composed of epithelioid cells, but not arranged concentrically, and sometimes we have found the core of granular elements, *i. e.* lymphoid cells. The part beneath the papillæ shows epithelioid cells of more irregular shape and size, and forming together much more irregular figures than the sections of the papillæ; still there is always in epithelial cancer a structural configuration of the elements into tubes and knots very characteristic. There may be abortive hairs or gland-tubes or lymphatic channels full of cancer-cells; it is here in the subpapillary part of an epithelial cancer that we find the laminated corpuscles which are so characteristic of this kind of cancer. These appear as gatherings of epithelial cells in layers tightly packed around a circular or oval central space, the whole very like the section of an onion or like a bird's nest.

We also meet occasionally, though very rarely, with diffused primary scirrhous cancer of the skin; this takes at first the form of a wide dusky, purple, brawny patch. It was thus in the case of a woman in the hospital, whose skin and subcutaneous tissue were indurated and brawny, and in some parts elevated into a number of small tumours, as shown in one of our models. The whole trunk being affected, it could scarcely be said with certainty how far the new tissue was fibroid and allied to keloid or scleriasis, or how far such a slow growth was carcinomatous, although, after some time, it was tolerably clear that it was of the latter kind, *carcinoma fibrosum* or *squirre en Cuirasse.* The woman dying some months after she left the hospital, a post-mortem examination revealed cancer, not only in the skin, but in the bones and other parts. Such is a good example of *scirrhous cancer.* An equally good one furnished another model, where we see a large dusky thickening on the front of a man's chest, which rapidly opened and proved to be scirrhous cancer. When a circumscribed prominent growth proceeds from the skin some distance as an isolated tumour, it generally is structurally a *medullary sarcoma*; if very vascular, it is what was formerly called *fungus hæmatodes.* We must again make the remark, which we have repeatedly made before, as to the difficulty of defining many forms of maladies, seeing that they pass by insensible degrees into one another, and thus we may conceive, that if a cancerous growth were very slow in its formation, and were of the fibrous kind, how much it would resemble keloid, and some other diseases already mentioned.

*Melanotic sarcoma* of the skin is a pigmented form of sarcoma generally composed of spindle-cells with more or less of an intercellular substance, that may or may not be fibrillated, but generally is so. The pigment is in the cells; sometimes it is very unequally distributed in them. It is very remarkable that often in these cases the pigmented character of the disease is determined by the previous presence of pigment in a mole. Thus, in one of our drawings there is a black tumour on the arm which originated in a mole, and the disease after excision soon returned in the axillary glands, as sarcoma combined with pigmentary matter. In some cases, however, the disease has no such evident origin. In the Museum is a piece of skin containing melanotic tubercles; and also the drawing of a man who was similarly covered, and who had the disease also in his internal organs.

*Nævus* we have already spoken of, and allied to it are mothers' marks and dark hairy patches, as seen in many preparations.

In connection with the integuments we meet also with fatty tumours, or *lipoma*, of which several large specimens are found on the shelves; also various forms of *fibroma* and *sarcoma*; the former composed of simple fibrous tissue, which appear to be altogether innocent; the latter composed of nucleated fibre, which often *recur* after removal. These have been called *fibro-plastic* or *recurrent fibroid.* We likewise meet with *myxoma*, a tumour of a softer consistence and gelatinous appearance. These all differ from cancer, in being often circumscribed, and not involving the neighbouring tissues. We have also met twice with pure *adenoma* of the skin and subcutaneous tissue, the structure being identical with that of the breast in lactation. One of these was partially charged with lime-salts.

We might here mention gouty concretions of urate of soda met with in the teguments under the name of tophi, chiefly in the ear, in the scrotum, or about the elbows, &c. The same deposit also invades the teguments from gouty joints.

**Parasites.**—Of animals that inhabit the skin the most common in our country is the so-called itch insect, the *Sarcoptes hominis*, or *Acarus scabiei.* This is not a true insect; it belongs to the class arachnida; the female has a rounded body, with eight legs, the four anterior being armed with suckers, and the four posterior with bristles; it has two pairs of mandibles armed with teeth, and is larger than the male. The female itch mites burrow in the rete mucosum, and lay their eggs as they work their way along, so that at the end of the burrow is the animal herself, while the eggs, in their order of juniority, lie in the tunnel behind her. Besides these tunnels or "cuniculi" there are usually eczematous vescicles due to irritation and the effects of scratching on the skin around.

Another creature of an allied nature is the *harvest bug*, *Leptus autumnalis*, of genus Trombidium, and undetermined species. Many of us may have been attacked by these tormenting creatures while walking through stubble fields in the autumn. By scraping the place which itches, the insect may be caught and put on a piece of white paper, when it appears as a minute red body.

In the West Indies a most tormenting creature is the *chigoe*, or *Pulex penetrans.* The female penetrates the skin, and there lays its eggs, producing in consequence an irritable sore. It is very like our common flea, but smaller.

The *Dracunculus medinensis*, or Guinea worm, is occasionally seen in the hospital, in sailors who have come from the coast of Africa and some other tropical climates. It is a long, thin, viviparous worm, belonging to the Nematoda. It measures several inches in length, as may be seen in any of our specimens, which were removed from the tegument of the body; the end protruding from the skin was secured, and the remainder of the worm gradually coiled out by slowly rolling round a piece of paper. It is supposed that they enter when very small through the ducts of the skin, taking advantage of opportunities when people are bathing, wading, &c., or they are found in the legs of those who have been occupied in boats where water has lain at the bottom, or in the backs of those who have been employed in carrying water; they come in the rainy season, and attain their full size in about a year.

We must here too mention the curious *fungus disease* of the foot, *Mycetoma Carteri*, for the first explanation of which we are indebted to Dr. Vandyke Carter, of Bombay, before whose observations the disease was supposed to be scrofulous, though its superficial characters had been described. In this affection the foot is found swollen, and having crater-like orifices on its surface, which lead into channels of about the size of a quill. These permeate the textures of the foot, traversing its tissues, bones as well as soft parts. In these channels is a fish-roe or poppy-seed-like substance, which may be of a light greyish colour, as in the variety called *madura foot*, or may be brown or black. In the latter case the microscope shows the presence of a fungus,—*Chionyphe Carteri.* There is no such fungus in the pale-coloured variety. In either case the substance has the form of clusters of little balls of an amorphous substance, each ball surrounded by a covering of radially placed, flexuous, cilium-like filaments, which Dr. Carter says are fatty crystals, but this being so their constancy, evenness, and regularity are very curious. The absence of the fungus in some cases is as certain as its presence in others, and the difference is not yet, we think, fully explained, but Dr. Carter considers the colourless form to be the remains of the fungus in an altered condition.

Lancereaux has reported some cases where the whole body, including the trunk and limbs, was covered with tumours which on examination were found to be *cysticercus cellulosæ.* They were situated in the subcutaneous tissue and muscles. He gives to the disease the name of *Ladrarie.*

## SUDORIPAROUS GLANDS AND DUCTS

We have not much to say in reference to these organs, but we may mention in connection with them the morbid condition known as *miliaria* and *sudamina.* Hebra distinguishes between these : sudamina, he says, are only swellings of the hair-sacs and mouths of the sebaceous follicles in cases of excessive heat and sweating. The name miliaria, on the other hand, he reserves for the same condition for which we employ it. Thus, in febrile affections a clear fluid collects beneath the cuticle in small transparent drops ; this fluid is often acid when tested, and so it differs from the inflammatory exudation in most vesicular diseases, as eczema, &c., where the fluid is alkaline. Hebra denies the acidity of miliary eruptions ; nevertheless in acute rheumatism especially it certainly is usually acid.

## SEBACEOUS FOLLICLES

The abnormal state of the sebaceous secretion gives rise to several important diseases of the skin. Thus, the secretion, being dry, plugs the mouth of the follicle, and then the follicle becomes distended with a mixture of secretion and epithelial scales drier than the natural secretion. This constitutes *comedo*, with which we are familiar as a little elevation with a black top, very common on young gentlemen's faces during the active sprouting of the beard. Where the skin is very thin, as in the eyelid and scrotum, the collection occurs in the form of a little white knot with the layer of skin running over it; this is called *milium.*

Where the sebaceous secretion is in excess, it may be spread over the skin in the form of scales, and is then called *seborrhœa.*

If an acute inflammatory product is formed around a comedo we then get *acne punctata;* or if the inflammation is severe around many follicles, especially in the dense tissue of the nose, *acne rosacea.* If, instead of acute inflammatory formation, a slow fibrous growth surrounds the follicle, increasing in the direction of least resistance, so as to rise off the surface and acquire a neck and at last a pedicle, then we have the pendulous sebaceous tumour of *molluscum contagiosum*, the contagious nature of which is, however, much disputed. Hebra denies it. Virchow admits it in a modified way, saying that where there is foul secretion from the sebaceous glands of one this is likely to irritate the sebaceous glands of another much in close contact, as a nurse child, for instance. We possess a model showing the usual example of such tumours around the mouth of a child. Mr. Hutchinson has published a similar example.

There seems, however, to be an actual growth in the gland, for a mass of cells are found in it, and amongst them some large ones which are peculiar and have been called mollusc bodies; occasionally also there has been present a central axis supposed to be the remains of the duct. Certain late investigations have thrown some doubt as to the origin of these tumours in the sebaceous glands, showing rather that they arise in the corium.

Sometimes the process by which comedones are formed reaches a new scale of magnitude, so that the cyst grows very large, as large as a walnut, an apple, a cocoa-nut. Such tumours are called *wens* or *sebaceous tumours.* Sometimes, as by Boerhaave and Sir Astley Cooper, the opening of the follicle is discovered, and the origin of it thus declared. Sometimes a fine cord has been found connecting the cyst with the skin and representing the obstructed duct. In by far the greater number of cases, however, the skin over the tumour is tense and shining, and no opening can be detected, so that these cysts are looked upon as new formations by some. Such cysts have a very thin wall of fibrous tissue from the corium; inside this is a thick compact layer of epidermic scales, with more or less fat between them. The youngest layers next to the cyst wall are best formed. The older are more broken down, and form a pultaceous mass in the centre, from the consistence of which Virchow adopts the old name atheroma for these tumours.

Sometimes the epithelial cells lie in layers, and between them are crystals of cholesterine, the whole forming a pearly mass called *cholesteatoma*, very similar to the tumour we mentioned as occurring sometimes in the membranes of the brain. Fine pale hairs are sometimes found in such cysts, as in similar cysts in other parts, mingled in the fat; and when these cysts are small, and mostly in young subjects, we may find the lining of the cyst to be much like skin, and to have hair-bulbs in it. Similar cysts bearing hair are found in the skin of some of the lower animals, the hair being like that natural to the particular creatures. Mr. Cock drew attention to the sprouting of growths from these large sacs after they have been opened, and a formation of warty growths in the interior of smaller epidermic cysts has been noticed by several observers. Sir James Paget points out the hereditary descent of these cysts in families.

Dr. Malherbe, of Nantes, has described a calcification of sebaceous tumours. There has been a growth of fibrous stroma from the interior of the cyst, which has in some instances become true bone; this had given rise to the term subcutaneous osteoma.

It is probable that some malignant ulcerations of the skin may have originated in cancers having their origin in the sebaceous glands.

## NAILS

*Ungues adunci*, or curved nails, in connection with phthisis, were observed as long ago as the time of Hippocrates. This condition is found on the toes as well as the fingers, and is not peculiar to consumption, as it is sometimes seen in heart disease, especially of the aortic valves, and sometimes in bronchitis. It reaches an extreme degree, too, in cases of empyema; it appears to depend, partly at least, on venous obstruction, especially when associated with hectic, but its causation is not quite satisfactorily explained. The lowest degree of this change shows itself in a thickened shiny state of the fold of skin over the root of the nail. Next the nail is thick and curved. In the extreme degrees the whole end of the finger is obviously thickened; but anatomical examination has not shown anything more than slight general hypertrophy of the tissues.

*Onychia* and *paronychia* are very painful diseases, accompanied by suppuration, affecting the root of the nail, and often leading to its loss; they are described by the surgeon. In *onychia maligna* the nail is discoloured, loose, and recurved, and rests on an angry-looking sore, while the end of the finger is clubbed and dusky with congestion.

Some cutaneous diseases implicate the nails and their roots, as well as the skin. This is the case sometimes in eczema, impetigo, or in the less acute inflammation of simple psoriasis. In pityriasis rubra they are usually shed with the whole epidermis. Syphilitic psoriasis, acquired or congenital, also occasionally, though rarely, affects the nails, causing their surfaces to be ragged and irregular, while the skin around the nail is inflamed and red.

Mr Hutchinson has drawn attention, not only to the state of the nails in syphilitic psoriasis, but to a state of them in congenital syphilis, which is very interesting in connection with his observations on the teeth in this condition. The nails are symmetrically affected; they become dry and brittle, and so are fissured and broken at their free edges; the superficial layers are alone affected. This condition is well represented in Mr. Hutchinson's plates in the Sydenham Society's 'Atlas.' There is, however, a more remarkable affection in the form of a chronic general onychitis, where the nails decay and fall off; they first become opaque and much thickened, and their substance is soft. The disease is due to an inflammation of the matrix, which is swollen and readily bleeds. This is also described in the Sydenham Society's 'Atlas,' and in Mr. Hutchinson's 'Catalogue of the Collection' he remarks on the very great symmetry of the disease, which shows its constitutional origin. He says it usually attacks children and young adults, and there is seldom any history of syphilis. In this our own observation agrees with his.

Dr. Fagge has observed ringworm affecting the nails; the nail was ribbed longitudinally, and its colour was mottled with dark brown; the texture brittle. Microscopic examination showed *Trichophyton tonsurans* in considerable quantity in the substance of the nail.

The handling of subjects injected with arsenical solution induces an inflammation of the bed of the nail, which may produce suppuration under it, an appearance illustrated by models in the Museum. On one occasion we saw a long continuance of the action of arsenic produce a warty growth in the seat of such inflammation, which caused great pain for many months.

There are other states of the nails which sometimes appear after illnesses, and are of great interest, as revealing the general nature of a change during some obscure constitutional affection. These are usually transverse notches across the nail, which, of course, move in the growth of the nail, so as to remain for months as evidence of what occurred.*

The nails, if neglected, may grow very long and curved in all directions, as in some curious specimens in our Museum (*Onychogryphosis*).

## HAIR

**Colour.**—Greyness of the hair depends on deficiency of pigment, or the presence of small vesicles of air in its shaft; the change begins, as is well known, on the temples, though in those prematurely grey this is not necessarily the case. Those who have investigated the subject give full credence to the stories related of the hair turning grey in a few hours from excessive grief.

*Atrophy* and *falling off* of the hair often occurs after febrile attacks, where, no doubt, the whole skin has been diseased. In various chronic maladies, as syphilis, &c., it partially falls off. Common baldness of elderly males has been plausibly ascribed to the wearing of tight hats compressing the vessels of the scalp. This natural baldness occurs at the crown of the head, and extends equally from this part; but there is a disease in which the hair disappears in circumscribed patches—this is called *alopecia areata.* There may be only one patch or more; it may even spread so as to remove the eyebrows, whiskers, &c.—in short, every hair from the body, producing *universal alopecia.* This was formerly thought by Willan and Bateman to be dependent upon a pustular disease, which they call porrigo decalvans; but such a baldness is by no means always preceded by any affection of the kind; and another theory is, that the decay of the hair is due to a vegetable parasite, which has received the name of the *Microsporon Audouini.* We think this is very doubtful; at least, we are among those observers who have never been able to discover a fungus in any of the hairs surrounding the bald patch, and therefore it has still to be satisfactorily shown what is the cause of the withering and falling off of the hairs in this manner. There is another affection of the hair, which we do not think is described in books, but about which we are sometimes consulted, and that is a *breaking of the hair.* Owing to the fracture, a white spot is produced; and if a number of these are present, as, for example, in the whisker, they have been mistaken for ova of pediculi, and alarmed the patient. If we examine one of these hairs, we find it broken in one or two places, and thus presenting a nodulated appearance; and if then examined by the microscope, the fibrous structure of the hair will be found to be broken up into a brush; we have seen interspersed a few dark granules, but we could never make out distinctly any sporules of fungi. Examples of what we mean are best seen in our drawings. It has since been called by Kaposi *trichorrhexis nodosa.* The growth of the hair varies much in health and disease. Curly stiff hair generally denotes health, while long straight hair, breaking at its ends, denotes a feeble and often phthisical subject. Such a distinction may be of value, with other signs, in assisting the medical man who has to examine a body as to the cause of death. If a patient has been long ill, and with a wasting disease, the hair is long and straight, both on the head and beard; but the condition is especially observable on the pubes, where the hair may be seen hanging in long straight tufts. In phthisis and some other wasting diseases there is often a disposition for the hair to grow, and thus the chest may have become recently covered with long straight hair.

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* See 'Lancet,' Jan., 1869; and 'Path. Trans.,' 1888.

**Vegetable Parasites.**—These have received a considerable share of attention, as it has been thought that some very important contagious diseases of the head are due to them. We have already mentioned one of these, the *Microsporon furfurans,* as the cause of pityriasis versicolor. These fungi are all more or less alike in being composed of simple cells and filaments; the cells are roundish or oval, and vary in shape and size in different species. If the cells are long and placed end to end, they constitute branching filaments, or a *mycelium*, as it is called; upon these is placed the receptacle, or *sporangium*, containing the reproductive *sporules*. The structure of these fungi is best brought out by the addition of a little alkali to the slide. The fungi are so much alike, indeed, that some observers, with Professor Hebra, believe they all have a common origin. This view is supported by the occurrence of favus in patches of tinea tonsurans. This association is shown in one of Hebra's plates, copied in the Sydenham Society's 'Atlas.' It has been suggested that the apparent tinea tonsurans was a variety of favus, but there is no evidence of this. Amongst the most important is the *Achorion Schönleinii*, which is the cause of the favus, or, as it is still called in some of our drawings and models, *porrigo lupinosa*, the name used for it by Willan and Bateman. We refer to the disease characterised by large yellow, raised, circumscribed cup-shaped masses growing on the surface of the skin, and more particularly the head, the skin between being little affected or unaffected. If these crusts be examined, they will be found surrounding the hairs, and composed of large oval sporules and a mycelium, as we have just mentioned. They may be found in the follicle, but do not necessarily destroy the hair. The *Trichophyton tonsurans* was first described by Gruby, as the vegetable fungus which causes ringworm or tinea tonsurans, or, according to our old nomenclature, *porrigo scutulata*. We allude to that disease of the scalp where there are branny, desquamating patches covered with short broken hair. The sporules are found to have entered the sheath of the hair, and crept upwards into the stem, so that it decays and breaks off short. The sporules are rounder and smaller than in the preceding variety, and distinct filaments are not so easily seen. It might be remarked that in this disease the parasitic growth gets into the fibrous structure of the hair itself, and not merely around it, as in favus. Dr. F. Taylor proved by examination of sections of skin that the hair itself was primarily affected and not the follicles. The *Microsporon Audouini* we have just mentioned is a fungus believed to cause the falling off of the hair in alopecia, but this we have never yet een. The *Microsporon mentagraphytes* is found in the sheath of the hairs of the skin, constituting the disease known as sycosis. This fungus is now considered identical with the *Achorion Schönleinii* of ordinary ringworm. We have frequently pulled out hairs and seen the sporules, but we cannot say that they exist in all forms of the disease, and that the pustular eruption met with is always due to this parasite. It is essentially a suppurative inflammation of the hair sacs of the beard. It is stated also that a species of trichophyton may be found in the peculiar disease of the hair occurring in Poland, where it becomes matted together, as seen in our specimens of *plica Polonica*.

We are not at all decided that these parasites actually constitute the disease, and that they are not accidental; indeed, we think a vesicular eruption may often be seen to precede their appearance.

**Animal Parasites.**—These are *pediculi*, or lice. The most common are the *Pediculus capitis* and *pubis*, the latter also called *phthirius*. The great difference between them is the length and shape, the one being long and the other of a squarer form. These are distinct species, and will not occupy the habitat of each other. Another species of phthirius affects the eyebrow. As lice are especially seen on sick people, it is thought a special genus attacks them, and this is called *Pediculus tabescentium;* but of the truth of this we cannot speak. It is also said that the creatures which one sees crawling on the clothes of beggars form a distinct genus, the *P. vestimentorum*. The eggs of lice are attached by a gummy substance to the hair, where they may be seen sticking in rows when the hairs are plucked out.

## THYMUS GLAND

Nothing is known of the function of this body, and almost as little of its pathology. It is said, sometimes, to be found *atrophied* in newborn children, showing that it must have undergone a change in fœtal life. Sometimes, in children, it is found large, and is said to be *hypertrophied;* in some cases we have seen the gland too large, but this has been due rather to a persistence of its earlier state, and not to any actual increase in size. It is thought, however, by some older German writers that such enlargement may cause pressure on the air-passages, and give rise to what they call *thymic asthma*, an opinion that would correspond with the view of others, who suppose that enlarged lymphatic glands compress the recurrent laryngeal nerves producing spasmodic croup. We have no experience of thymic asthma. Modern German writers, as Friedleben, have thrown much doubt upon it; Virchow holds it possible, and gives a case of fatal asthma in a child, wherein he found the thymus to weigh six and a half drachms. In later life the thymus should have nearly disappeared; but it has happened very curiously that a large thymus has been discovered in an adult with leuchæmia, and also once or twice without leuchæmia. Thus we met with a thymus gland weighing one ounce and a quarter in a man of thirty-two years, who died of purpura; his spleen was large as in leuchæmia. It is interesting to see how pathology thus agrees with anatomy, in allying the thymus with the other blood glands. Some interest was created by Dubois's observations of suppuration of the thymus in visceral syphilis; these at first received partial confirmation, but of late have fallen into some neglect, and we think doubt. We have seen the usual milky secretion in the thymus in syphilitic infants, but we have not seen pus.

Abscess is a rare result of syphilis. Occasionally, tubercular disease has been found in the thymus, and, we believe, cancer. Among our preparations we have one, of enlarged thymus, and another, which is called cystic; but it is doubtful whether it is thymus at all.

## THYROID GLAND

There is an *acute enlargement* of the thyroid sometimes met with. It usually occurs in young persons and in connection with rheumatism. It lasts for a few days and is attended with much fever. The French have described fatal cases of the disorder.

The most common form of affection of the body is the *chronic enlargement*, as seen in *bronchocele* or goître, when it often reaches an immense size, but less degrees of its enlargement are constantly met with on the post-mortem table. Sometimes, these enlargements appear to be *simple hypertrophies*, the tissue being close and uniform; but very often the size is due to a jelly-like or colloid matter being formed in the interior of the organ, and sometimes the loculi dilate into large cysts filled with this gummy substance. The latter, when in great quantity, is fluid, but sometimes is as firm as wax. Several specimens are on our shelves of these *hypertrophied* and *cystic* thyroid bodies. Besides this general change, very often distinct and isolated cysts are formed, growing from one lobe or the other, and attain a very great size; and sometimes the walls of these *ossify*, as seen in our specimens. Knowing that such are not uncommon, it is not surprising that an hydatid cyst which Mr. Cock removed some years ago from the thyroid should have been looked upon as part of a bronchocele. We have seen this body involved in inflammatory and suppurative processes in the neck, but not, we think, primarily so. Tubercle we have never met with, and *cancer* but rarely. It is generally agreed that cancer of the thyroid is very rare. Walshe estimates the proportion of thyroid to other cancers as 1 to 1000; we have found it only on three or four occasions, and in all but two the fact of the neighbouring lymphatic glands being implicated, made it questionable whether the disease had not commenced in them.

We have a specimen of cancer of the thyroid from a man, aged sixty, who had cancer of the lymphatic glands, lungs and liver. The enlargement had existed for ten years and therefore it was thought that the cancer was engrafted on an old bronchocele. Another specimen of cancer is from a child who was under Dr. Fagge in the Evelina Hospital.

A few examples have been published of a very remarkable form of disease, looking like bronchocele with secondary growths. Mr. Warrington Haward had a case of enlarged thyroid which was apparently an ordinary bronchocele. Growths occurred in other parts, especially the bones, resembling this in every respect; in the skull, scapula, ilium, and spine, as well as nodules in the lungs. They had a basement of connective tissue supporting in its meshes numerous alveoli lined by cells, and resembled, indeed, thyroid tissue. Mr. Henry Morris had a remarkable case of a vascular tumour of the head; there were growths in other bones. These were not like ordinary nævi but in structure very like the thyroid body. This was the opinion of Cornil who examined them. A similar case is recorded by Langenbeck.

We must here consider the anatomical state of the thyroid in the curious condition known as exophthalmic goître, or Graves's disease, or, more rightly, Basedow's disease, since he first, in 1840, recognised it as a special cachexia, and described it so fully that little has since been added. In this condition, when fully developed, the eyes are prominent, the heart irritable, and the thyroid large; we have already spoken of the healthy state of the eyeball. The thyroid equally shows no essential change; it is generally very vascular, and it varies in size. In thirteen cases collected by Virchow, six had the tissue normal, but hypertrophied, and the vessels large; in the other seven there were local changes, thus one had some gelatinous degeneration, three had cystic changes, another had hæmorrhage into the tissue in spots, and another a cartilaginous patch. Of three examples we have inspected, one had the gland normal in texture but large, the other two had slight local changes in the form of tumours of thyroid tissue. Thus, there is no known post-mortem condition of the thyroid gland proper to Basedow's disease. During life we generally hear curious loud vascular murmurs in the gland and neighbouring part of the neck. We have specimens of the exophthalmic goître in the Museum.

In the disease known as *adult cretinism* or *myxœdema* the thyroid has been found completely atrophied or sometimes altogether wanting. The patient's hebetude is supposed to be intimately associated with the functions of this gland, for when the organ has been removed from the presence of a tumour, the subject of it has in course of time shown extreme mental weakness. In a case of myxœdema occurring at St. Thomas's the thyroid was enlarged and occupied by hard cancer.

In the *sporadic cretinism* of children described by Dr. Fagge, the thyroid was usually found altogether wanting, but there was a mass of fat situated over each clavicle. In some cases the thyroid body was present, but the subject of disease. We shall again allude to it under "General Diseases."

# DISEASES OF THE RESPIRATORY ORGANS

## AIR-PASSAGES

These are divided into the LARYNX, TRACHEA, and BRONCHIAL TUBES. In many cases we shall have to speak of these parts separately, but, as they are so often the subject of disease at the same time, we shall prefer taking the morbid conditions one by one; and then, if need be, show how each individual part is especially affected.

**Dilatation or Bronchiectasis.**—A dilatation of the *trachea* has been mentioned by Rokitansky as occurring in its posterior part in old people, but we have hitherto failed to find it; and we shall therefore pass on to *dilatation of the bronchi.* This has been divided into various kinds, but we will only mention the two principal varieties, as they, for the most part, correspond to two very different pathological conditions. One is a cylindrical and uniform dilatation, and the other a saccular; the first is usually associated with a simply collapsed condition of the adjacent lung tissue, and is a primary affection; while the latter is connected with a structural change in the parenchyma, and is mostly a secondary process.

To commence with the saccular variety—we mean by this term a condition of tube where a tendency to sacculation is approached, and not where distinct sacs are formed, which is rarely the case. Such dilated tubes have long been observed in the upper lobes of the lung in connection with chronic pneumonia, and in the similar indurated condition of tissue in phthisis; in these cases, where a vomica has been suspected, but a mere dilatation of a tube found, it was formerly thought that the latter had preceded the induration of the tissue; but some years ago the opposite theory was propounded by Corrigan, that the disease of the lung substance preceded, and caused the expansion of the tubes. He studied the subject especially in those cases where the lung was diseased throughout, and, being converted into fibrous tissue, was styled *cirrhosis;* but the same causation holds good where portions only of the lung are affected, as before said. This theory is that, owing to the chronic change in the lung, and its conversion into a new fibrous tissue, a contraction ensues, and that as a consequence the bronchial tubes are drawn open; just as during the same contractile process, the walls of the chest are drawn in. Thus, in certain specimens of cirrhosis the lung tissue is quite gone, and its place occupied by a dense fibrous structure, and through it are seen large irregular spaces which have orignated in the expansion of bronchial tubes; the same thing may be seen in most cases of phthisical lungs, if the disease be of any standing. In such cases it is often asked, whether the cavity or hollow space is one formed in the substance of the lung, or is a dilated tube; but it is very difficult to decide with certainty, and, indeed, it is often a mere question of words, for the cavity becomes so altered by dilatation that the mere continuity with the tube is not sufficient to prove the point; for, supposing in the first instance a tube should dilate, it afterwards increases at the expense of the pulmonary structure, and in the meantime the original character of the bronchus is lost; the cavity having a smooth wall but no epithelial lining.

The other form, or general dilatation of the tubes, although associated with an atrophied state of the pulmonary tissue, has its origin in a bronchitis. It is found in long-standing cases of this disease, especially in children, where it has succeeded to a hooping-cough, as may be seen in one of our specimens, which will explain the nature of the affection. This lung came from a boy who had suffered from severe bronchitis (or asthma, as it is popularly called) for many years, and the physical signs suggested cavities at the bases of the lungs. These organs, however, were in the condition now seen: the primary and secondary divisions of the bronchi are natural until the lower lobe of the lung is reached, when the tubes become expanded, and occupy the greater portions of its lower part like the fingers of a glove; they get wider and wider until they reach the bottom of the lung, when they abruptly end, being there as large as the primary branches of the bronchi themselves; other parts of the lobe show large spaces or cavities, which are sections of the tubes.

There is an old specimen of Dr. Bright's in our Museum taken from a young man aged twenty-seven. The lung is occupied with dilatations of tubes forming large cavities and filled with cretaceous matter. The walls are thick, but the lining thin and highly vascular. This vascularity has suggested a close relationship between the tubes and pulmonary tissue, and that the process of dilatation is of a more active kind than is generally supposed.

In these cases the mucous membrane is generally red, swollen, and villous, and covered with secretion, whilst the walls are thin; in the other or saccular variety, when in connection with cirrhosed lung, the walls are thickened from being involved in the fibrous induration around them, the mucous membrane is thickened, and the longitudinal and circular fibres are hypertrophied; the circular passing as distinct ridges around the tubes, but in some saccular forms the muscular and elastic fibres have almost disappeared.

It is evident that neither form of dilatation can occur without an atrophy of the intervening tissue; but the relation which these two conditions bear to one another is still a subject of controversy. In the first-mentioned variety, where there has been a distinct history of chronic pneumonia or pleurisy, we must regard the change in the parenchyma as the starting-point in the process, but in the second variety of general dilatation following a bronchitis, it is probable that the inflammation of the tubes has originated it. There are pathologists, however, who, following Laennec, maintain that under all circumstances the primary morbid state is in the tubes, that in one case a simple dilatation, with collapse of the parenchyma, results, whilst in the other a lobular catarrhal pneumonia following the bronchitis, and leading to solidification and contraction of the lung, brings about dilatation. That the inspiratory efforts, acting on a softened tube and inflamed lung, are sufficient, and that a bronchitis can produce dilatation is shown by a case which occurred here in the person of a lad who died of bronchitis nine days after an accident, and in whose lungs the tubes were found greatly distended; the parenchyma was healthy, and the condition found was no doubt a recoverable one. In cases of longer duration the intervening tissue may be found of a dark colour, tough, and airless, and the air having been squeezed out, the lung has become carnified. As the whole lung must occupy a certain space in the chest, one portion of it cannot contract without another expanding, and *vice versâ*. It is therefore probable that the two processes of dilatation of the tubes and contraction of the tissue may be in progress at the same time; a bronchitis and blocking of the smaller tubes would prevent an ingress of air into the cells and thus cause a collapse of the tissue, and this might again lead to an expansion of the tubes. It is questionable whether the force of air during inspiration would be sufficient to cause a permanent dilatation without the intervention of the collapsing process, and it is therefore probable that the two conditions which are so often found together and necessarily associated, a dilatation of the tubes and a shrinking of lung tissue, are in progress at the same time. In the case of a little girl who had been cyanotic since infancy, and died dropsical, the tubes in the lower lobes reaching to the surface of the lung were as large as the little finger, whilst nothing but atrophied pulmonary tissue was visible between them. Here there had probably been a bronchitis, followed by collapse and subsequent complete atrophy of the tissue. In a case of cancer of lung which had penetrated along the bronchial tubes the latter were extremely dilated.

The cases of saccular bronchiectasis most difficult to understand are those where a mass of tubes are immensely distended and in close approximation without a trace of healthy parenchyma between them. In the case of a woman, without any history of a general bronchitis, the upper lobe consisted of nothing but a congeries of immensely dilated tubes; and several other cases we have met with accidentally on the post-mortem table where the condition was purely local and with no symptoms. The lungs have a honeycombed appearance, and consist of a congeries of loculi separated by extremely thin walls, reminding one of similar cysts in the liver and kidney. It is a kind of emphysema of the bronchial tubes. The sacs sometimes communicate with one another.

**Contraction.**—From *pressure* without; thus the trachea and bronchi are constantly found compressed and flattened by aneurysm of the aorta, and more rarely by other tumours. Mr. Wilkinson King wrote a paper in our 'Reports' showing that the enlargement of the left auricle, which occurs in some forms of cardiac disease, exerted a pressure on the left bronchus. The preparations are in the Museum, but the fact mentioned is not very obvious. Constriction may arise from *disease within*, as from contraction of an ulcer, illustrated by specimens of trachea, and contracted bronchus, where the tubes are diminished to half their width; the former appearing as if a tight ligature had been placed around them. In all these cases the patients were *syphilitic*, and there could be no doubt that this was the character of the disease; such, also, has been the case in the few other instances we have seen. Such examples may be known during life, not only by the physical signs of the contraction, but by the occasional discharge of portions of the cartilaginous rings during coughing.

In a case which occurred more recently the larynx was healthy, but immediately below it a contraction commenced, and extended throughout the trachea, the surface was ulcerated, and was mainly composed of cicatricial tissue; in some places the tracheal rings were exposed, and at one spot was an opening which passed into the aorta; this caused death by hæmorrhage; at the middle portion of the trachea the internal surfaces actually touched from the extreme narrowing of the tube. A case is recorded where a syphilitic ulcer of the right bronchus opened into the pulmonary artery, causing a fatal hæmorrhage. Not long ago a man came into the Clinical ward with threatening suffocation; tracheotomy was attempted, but the cannula could not be thrust into the trachea from its extreme narrowing. In these cases it is probable that the cause was syphilitic, but this need not always be assumed, for it is possible for a tracheitis and bronchitis with formation of a dense fibrous tissue to have its origin in other causes, although this is extremely rare. It is remarkable how little ill consequence follows the operation of tracheotomy, and indeed how slight is the trace which is left. In a case of a child Mr. Jacobson operated on a good deal of cicatricial tissue with narrowing was found fourteen months afterwards, but then the child had swallowed chloride of zinc.

Dr. Lees exhibited at the Pathological Society the larynx of an infant who had during life crowing respiration; the organ had the epiglottis folded upon itself, and this condition he regarded as congenital. Dr. Mackenzie also described a case where the anterior parts of the cords were united, and there is a specimen in our Museum showing the left sacculus laryngis everted. There were no symptoms.

**Congestion and Hyperæmia.**—As we have already mentioned in speaking of other organs, it is very difficult to say where congestion ends and inflammation begins, and this is especially the case in the bronchial tubes; indeed we believe here, as in some other instances, the one passes into the other. This is especially seen in heart disease, where the state of the mucous membrane appears to arise in the first place simply from congestion, but yet in course of time assumes a character which cannot be called by any other name than subacute inflammation. Rarely, except in most acute forms of bronchitis, is the lining membrane of the tubes seen to be so red as in cardiac disease; it is often of the most intense character, and at the same time the tubes are full of a purulent mucous secretion. A similar condition will be found often in the stomach and intestines, as we shall hereafter mention. It appears as if, owing to the tension of the blood-vessels in heart obstruction, not only is a serum poured out from the serous surfaces, but an analogous mucous secretion from the mucous. This cardiac congestion of the tubes is only an extreme form of what is constantly met with in less degrees in other diseases, where there is obstruction in the circulation, and the lungs are gorged; as, for example, in disease or injury of the spine, productive of paralysis of the chest. The congestion may be passive, as in fever and in blood diseases; also in a large number of other cases which we have not time to mention, as in various forms of strangulation and drowning: here the redness is often considerable.

**Inflammation of the Air-Passages.**—The simplest form is called *catarrhal*, where the mucous membrane is inflamed, and a fluid secretion pours from its surface. It may arise as an idiopathic affection, though it is constantly occurring in connection with other diseases. It is difficult to tell how long it has existed from mere inspection of the tubes, or from the amount of secretion poured out; for this may be very excessive without any great inflammation. The inflammation of the mucous membrane is known, in the first place, by the secretion found on its surface; this, if it be of a muco-purulent character, or apparently altogether purulent, shows that the inflammation is violent; but it is rather from the character of the membrane itself that the disease is recognised, for not only is there an exudation from the surface, but also into the membrane, and thus it has a swollen appearance, and the surface is velvety or granular, besides being highly vascular.

When the *larynx* is affected the disease is styled *acute catarrhal laryngitis*, the urgency and importance of which are in proportion as the glottis and upper part of the tube are affected. In fatal cases, where an opportunity is presented of seeing the organ, we generally find the epiglottis of an intense red, and so swollen that it is almost closed, the epiglottis also involved, and the parts below showing ordinary signs of inflammation, with secretion on the surface, and infiltration of the mucous and submucous tissue. The microscope displays an ordinary muco-purulent secretion with epithelial cells. In many cases laryngitis arises apparently from cold. In others it is the principal symptom in measles, and sometimes requires tracheotomy. The inflammation may be membranous, but it is usually not so. We should also remark that we by no means regard tracheotomy as a harmless operation. We have repeatedly seen cases in which, the cause requiring it having at once been removed, as a foreign body, the child has nevertheless succumbed to acute inflammation of the air-passages, beginning at the place of incision. We have seen numerous instances of most intense inflammation of the glottis arise from simple local causes in children who had swallowed boiling water and acrid poisons; in these cases the glottis and epiglottis were acutely inflamed and swollen, and produced suffocation. We would here state that suffocation does not necessarily indicate a complete choking of the passage, as the closure is due in part to a spasm arising from the irritation, and perhaps more commonly to a paralysis of the glottis, for it must be remembered that when a muscle is inflamed it is paralysed, and thus the larynx closes during inspiration. Such acute inflammation is often the sequel of chronic disease, as syphilitic laryngitis, &c.; and in some cases death is produced by closure of the passage, where, on examination, no redness is present, but merely swelling; and this condition is styled *œdema glottidis.* It is often a mere dropsy, or exudation of serum into the cellular structure, and is thus met with in Bright's disease. We may see in our specimens, and very often better still in the post-mortem room, how the glottis is swollen and almost closed by this œdema, and sometimes the epiglottis is affected at the same time. It seems, however, to be sometimes a primary affection and sometimes associated with erysipelas.

Another form of laryngitis which would scarcely ever come under the notice of the morbid anatomist is *glandular* and *granular laryngitis,* where the follicles and glands of the mucous membrane are mainly involved. It is the peculiar affection met with mostly in singers and clergymen, and will be mentioned again under "Pharynx."

A much more formidable disease is the erysipelatous or *suppurative laryngitis* in which a purulent secretion rapidly takes place in all the tissues of the larynx and surrounding parts. It is often found in connection with an erysipelas of the neck, when a sore-throat and laryngitis is found accompanying it; in the course of a few days the patient dies suffocated, with a great swelling of the tissues of the neck. The cellular tissue is found infiltrated with a sero-purulent fluid, the muscles themselves may contain small deposits of pus, and suppuration may be found in the muscles of the pharynx, in the tonsils, in the submucous tissue of the larynx, and in the epiglottis itself.

This is a form of disease which has long been recognised, and is alluded to in a paper on Pyæmia, to be found the 'Guy's Hospital

Reports' for the year 1861, where several cases are mentioned of a diffused suppuration in the neck involving the larynx, and often causing œdema of the glottis. Mr. Hilton was of opinion that the dyspnœa was caused in many cases by the implication of the pneumogastric nerve in the inflammatory products of the neck. This long-known and well-recognised disease appears to be the same which Niemeyer styles "Angina Ludovici."

If the inflammation should affect the trachea in an especial manner, the affection would be called *tracheitis;* but we have never seen this tube solely affected; and we shall therefore pass on to the bronchi, and speak of *bronchitis.* This is characterised, also, by the state of the membrane and the secretion from it; as a rule the more purulent the matter thrown out, the more acute is the disease, and this is generally also in proportion as the smaller tubes are involved; and thus one should open carefully the ramifications of the bronchi, for more will be found there to account for the symptoms and death than in the larger passages. They may sometimes be discovered to be completely filled with very tenacious purulent mucus, and on making a section of the pulmonary tissue, small drops of thick matter may ooze out of the minute tubes: a most important morbid condition, which should always be looked for. At the same time the mucous membrane presents a red, swollen appearance, with its healthy character almost gone—looking, indeed, more like a bleeding fleshy surface; and on section these tubes appear almost closed from the increased thickness of the mucous membrane. If the tubes are distended, as we just now mentioned, the original character of their walls appears quite destroyed, but often they are thickened with a great hypertrophy of the elastic and muscular coats, the longitudinal fibres being very prominent.

When a large bronchial tube with its branches has alone undergone great thickening, we believe the cause has been syphilitic.

It must be remembered that the mucous membrane of the air-passages is covered with a cylindrical and ciliated epithelium, and in which are mucous follicles. The secretion thrown off will be found to contain these cells but more especially lymph-cells, which are being constantly produced when the former are shed. Clinically we separate ordinary bronchitis from capillary bronchitis; the latter being more severe and apt to run on so as to invade the lung tissue. We speak of the capillary tubes as the smallest tubes where all cartilage is absent, and there are no distinct muscular fibres.

*Plastic* or *croupous inflammation* affects the air-passages, the exudation being membranous instead of muco-purulent. The most important affection of this class is that known under the name of *cynanche trachealis*, or *croup*, a name which appears to denote that the windpipe is alone affected, but in most fatal cases which we examine the whole of the air-passages have been inflamed; but this may be a reason for supposing that, in instances of recovery from croup, so extensive an inflammation could not have existed, and certainly there is no reason to suppose that a *bronchitis* must necessarily accompany an inflammation of the main passages; but we think there is every reason to believe, both from symptoms and necroscopic examination, that the *larynx* is always involved with the trachea. Our opinion is, that in croup the larynx and trachea are always affected, and in more severe cases the bronchi also.

It may be observed that the term croup is not used in conformity with a distinct pathological condition, but as indicative of certain symptoms denoting a closure of the glottis, and, therefore, may include several affections. Thus, besides a spasmodic croup there may exist laryngitis of a simply catarrhal character, and another form where a false membrane is secreted. To both of these the term croup would be applicable during the life of the patient, but it is only to the membranous variety that the term true croup could be used were the condition actually known. Thus it is that the term "croupous" is made to apply to all membranous inflammations of the mucous membranes.

In a case, then, of well-marked croup, we find a membrane covering the larynx and trachea, beginning often on the under surface of the epiglottis, and extending downwards to the bifurcation of the trachea. It has but slight adhesions, and may be removed entire as a hollow tube, as may be seen in our specimens. In most cases where we have an opportunity of examining the body after death we find the exudation does not end here, but extends into the bronchi; the membranous character, however, often ceases at the bifurcation, and is there changed into a corpuscular or purulent secretion. In cases which recover the bronchial tubes are but slightly, if at all, affected. The cast, when examined, is found to consist of simple fibrillated lymph holding corpuscles in it; the mucous membrane beneath is generally red from vascular injection, but often this stage has passed away, and the membrane is pale. After removal during life, the membrane will often again rapidly form, and if not, the mucous surface soon returns to its healthy condition. Sometimes the lymph, instead of forming a complete mould, occurs in patches.

Until late years we have been content to speak of these two forms of inflammation affecting the air-passages, the *catarrhal* and the *croupous*, but now it is necessary to allude to a third styled the *diphtheritic*. Since diphtheria has been rife amongst us not only is there observed the more common affection in which the throat is covered with a false membrane, but there is another in which the membrane may extend through the air-passages after the manner of croup. This affection has created a division amongst physicians as to its nature. Is it peculiar and a manifestation of a constitutional disease, or is it in reality the same as the long-known croup? In other words are there two varieties or only one, of membranous inflammation of the air-passages? Is there a purely local affection known as the oldfashioned croup, and is there another of more recent importation associated with a constitutional disease? Or, on the other hand, has the well-known croup been the same disease as diphtheria, but appearing until late years as a milder and more localised affection? The most eminent men in the profession may be found holding different views. Those who maintain that there can be a simple membranous inflammation of the mucous membrane apart from diphtheria, or any contagious constitutional affection, would refer not only to ordinary croup, but to such cases as have occurred here where children, after swallowing boiling water or sucking in a nutshell or other foreign substance, have had a croupous inflammation of the larynx, trachea, and bronchi, and to other cases, as of phthisis, where patches of membrane may frequently be found on the interior of the windpipe, and actually covering a tuberculous ulceration. It is also maintained that the ordinary croupous membrane differs from the diphtheritic in being thinner, looser, softer, easily separable, leaving the surface healthy, whilst the diphtheritic is much tougher, like wash-leather, is a more complete cast, more densely fibrillated and more firmly adherent, and leaving a bleeding surface beneath when removed. Both these membranes, however, when placed under the microscope are much alike, showing a fibrillated tissue containing leucocytes and blood-corpuscles, and, generally speaking, also bacteria and micrococci. If these infective parasites be always present, and if it is to their presence that the irritative inflammative production of a membrane is due, it can be easily understood how other irritants, as boiling water or mechanical substances, can do the same. All these cases then of membranous inflammation are, to a certain extent, parallel. It may therefore very well be believed, since cold and measles produce only a simple laryngitis, and irritants of whatever nature may set up another process in which the production of fibrinous membrane is the result, that in all cases where membranous croup arises spontaneously diphtheria may be regarded as the cause. The differences mentioned between the case of the well-marked diphtheritic membrane on the throat and that occurring in croup, in the fact of the former being more adherent and the latter much looser, may not be of much value, as the explanation may lie in the anatomical difference between the tougher epithelial surface of the throat and the softer epithelium of the bronchi.

Having spoken of the trachea, we will now pass to the same affection, or *plastic exudation, in the bronchial tubes.* This, as before said, may be merely a part of a general croupous inflammation of the air-passages, but, as a rule, the membrane ends with the trachea, and then gradually passes into a softer or purulent secretion. Sometimes, however, it continues throughout. When the tubes are alone affected the disease has been called *plastic bronchitis;* this we rarely meet with in the dead body, but we occasionally see it in persons suffering from bronchial complaints, when the casts have been thrown up. These take on the form of the tubes, present an arborescent appearance, and then are expectorated, as may be seen in our preparations. Instead of the secretion being corpuscular and in the form of purulent mucus, it is lymph moulded into the shape of the air-passages and their minute ramifications.

These casts are usually hollow, and sometimes, when carefully examined, may be found to be composed of several layers one within the other. The same question arises here as in the similar affection of the larynx and trachea, whether this plastic bronchitis is merely one variety of mucous inflammation, or whether it denotes the existence of a specific or special cause of the nature of diphtheria. When the same patient has had several attacks it cannot be regarded as diphtheritic, but as a peculiar form of bronchitis.

The *solid casts* which are occasionally expectorated consist, in all probability, of fibrinous material derived from decolorised blood. We sometimes find, when examining a hepatised lung on the post-mortem table that the tubes are full of inflammatory exudation. This seems to be of the same character as the albuminous matter secreted into the parenchyma; it is solid throughout and gelatinous in consistence. There is no evidence of any inflammation or irritation of the bronchial membrane. It is not uncommonly found in the smaller tubes, but very exceptionally are the larger tubes filled with this exudative material.

*Results of inflammation and chronic changes.*—Pustules are sometimes met with in smallpox. In one case the whole of the larynx and trachea, as well as the œsophagus, was covered with smallpox pustules and shreds of false membrane.

Thickening or ankylosis of the crico-arytenoid joints is spoken of in connection with impaired mobility of the vocal cords.

One of the most striking changes in the larynx is the wasting of the muscles from pressure on the recurrent laryngeal nerve. The difference is most marked between the red, plump crico-arytenoid posticus muscle on one side and its yellow shrivelled fellow-muscle on the opposite side.

**Ulceration.**—It is remarkable that simple catarrhal inflammation rarely passes into ulceration, and therefore we must remember that this is not one of the results of bronchitis. The affection lasts for years, but without any breach of surface; and although this may occur to a slight extent at the upper part of the larynx, as the result of various forms of inflammation, yet in the trachea and bronchia simple ulceration is an event rarely witnessed. When it does occur it is associated with tuberculous phthisis in by far the majority of cases. As a rule, then, idiopathic ulceration does not often happen; for in the most severe inflammations, when the secretion or lymph is removed, the mucous surface will be found entire, and if found abraded, it has generally arisen from the imbibition of an irritating fluid. When met with, it is found in children in connection with diphtheria or other inflammation of larynx. Occasionally a most extensive ulceration is found with exposure of the cartilaginous rings, but this is generally regarded as syphilitic.

We may remember that ulceration of the larynx is common, that of the trachea less so, and ulceration of the bronchi rare. The most common cause of ulceration of the air-passage is tubercular; the syphilitic may be observed in the form of scars, and simple ulceration is rare. We must not forget, in cases of tracheotomy, that a patch of ulceration in the trachea is often caused by the cannula. In one case the spread of ulceration from the wound opened the innominate artery; then there is ulceration from aneurysm and morbid growths; also the very remarkable condition sometimes seen of a simple ulceration between the œsophagus and trachea.

**Aphthous.**—This is the simplest form of ulceration, and is found associated with the same disease in the mouth. It is characterised by a few very small follicular ulcers being met with in the larynx, and is sometimes seen in phthisis.

**Tuberculous Disease and Ulceration** is met with in most cases of phthisis that have had any duration. In an early stage minute white points of corpuscular formation may be seen scattered through the mucous membrane; after this a softening takes place, and minute ulcers are formed; at a subsequent period these run together, and, fresh matter being produced, a large ulcer results as an uneven surface, with white points of deposit on the base. It seems a matter of doubt whether this should be called true tubercle, but some of the larger deposits, as at the posterior part of the vocal cords, appear to be unquestionable tubercle. In early cases tubercles may certainly be found, but in more advanced cases they may not be apparent, the microscope being required to discover them on the edge of the ulcer. A case has been recorded where a tuberculous mass the size of a pea was found.

Sometimes the whole larynx and trachea may be thus affected, but generally the ulceration is in patches; and next to the larynx a favourite place is the trachea, just above the bifurcation, and then not unusually the ulceration extends down into the bronchi and their branches, and more especially between the rings. Its principal seat, however, is the glottis, and neighbourhood of the vocal cords; and here sometimes disease may be found, whilst all other parts are healthy. An ulcer may be seen at the posterior attachment of the vocal cord, at the base of the arytenoid cartilage; whereby the latter is exposed, and the joint opened. The ulceration then creeps along in the course of the cords until these are wholly destroyed; and in some cases the disease extends along the epiglottis, and most especially the inner surface, so that its edges are found eaten away; this produces the most troublesome symptom towards the finale of phthisis, and especially if it should extend still further, as is sometimes the case, into the pharynx; then we find not only the interior but the top of the larynx destroyed, and the outer or pharyngeal surface also involved, causing sometimes a suspicion of cancer. As it is difficult to see the primary seat, the parts first observed ulcerated during life are the vocal cords. This is the disease known as laryngeal phthisis; but it is a bad name, as it perpetuates the error that tubercular disease may be located in the larynx alone, as one of the forms of phthisis; probably there is no such disease apart from a similar affection of the lungs themselves.

As the laryngeal affection is so often secondary, it has been suggested that it has been set up by the passage over it of irritating secretion from the lungs, especially, as it is said, the tube of the most diseased lung is most ulcerated. The opinion is strengthened by the fact of bacilli constantly passing over it. Rarely, however, there does seem to be an acute miliary tuberculosis of the mucous membrane concurrent with a similar affection of the lung. In such a case the term laryngeal phthisis would be appropriate. It must not be forgotten that aphonia sometimes occurs in phthisis without any laryngeal disease as if due to nerve cause.

**Syphilitic Disease of the Larynx and Air-Passages.**—In the tuberculous disease just mentioned, apart from the small amount of adventitious scrofulous deposit, the affection is characterised by extensive ulceration, whereas in the syphilitic form the peculiarity is the thickening and induration owing to a formation of fibrous tissue. The difficulty is in distinguishing between a syphilitic and a simple inflammatory form of disease; but we believe the majority of cases of *chronic laryngitis* met with are syphilitic, and the more likely is this to be the case when there is a large amount of fibrous deposit present. In constitutional syphilis the mucous membrane is first merely injected, then mucous patches may be observed, and finally the production of lymph, which may subsequently become a tough fibrous tissue; this may be seen in periosteal nodes, as well as in the same formations in other parts; and thus in the larynx there may be found sometimes nothing more than a mass of fibrous tissue developed in the glottis, and almost closing it, as in the specimen on our shelves; in other cases we find, with this extreme thickening, also the epiglottis thickened and hardened; or this condition may extend down the larynx as far as the trachea; or the whole organ may be indurated throughout, and even sometimes the cellular tissue externally with the adjacent small lymphatic glands all matted together, and implicated in the process. With this induration there is generally more or less destruction of the parts, and in most cases, no doubt, an ulcerative process has accompanied the induration and contraction; and thus the inner surface has either lost its mucous membrane, or presents a cicatriform appearance; the vocal cords may be altogether destroyed, the epiglottis also partly or wholly gone, or presenting a contracted, withered appearance. Sometimes, if the ulceration is considerable, the whole of the inner surface of the larynx presents a shaggy or flocculent aspect, and occasionally the ulceration is continuous over the glottis, with an ulcer of the pharynx; in such a case the question may arise as to the original site of the disease; but, as both these parts may be independently affected, it is possible that the disease has progressed in both parts simultaneously. The earlier stages of syphilitic inflammation of the larynx would seldom be met with on the post-mortem table; but during life we find at the outset of constitutional symptoms a simple redness with secretion as in the catarrhal form; to be followed, perhaps, by raised patches on the mucous surface, which are sometimes designated condylomata.

Since special attention has been given to the internal syphilitic affections the peculiarities of the more chronic laryngeal diseases have been observed, and a real gummous perichondritis of trachea and laryngeal cartilage has been several times noticed. A homogeneous material may be found surrounding the air-passages, and involving their coats, and spreading around the œsophagus and neighbouring tissues; we have seen the œsophagus and trachea laid open, with fatal hæmorrhage, and sometimes, by the softening of the syphilitic material, an external abscess, and necrosis of cartilage. The mucous membrane is thickened, puckered, or ulcerated.

Other parts of the air-passages may be affected as well as the larynx, as seen in one of our specimens, where the lower part of *trachea* is very much thickened, and its surface ulcerated; and in another preparation of contracted *bronchus* arising from an ulcer, the nature of the disease was clear, in the fact of the patient dying of syphilitic laryngitis; the contracted trachea also had the same origin. As we before mentioned, in some of these cases of ulceration of the trachea the rings are laid bare, and sometimes become detached during life, if the patient recovers.

The syphilitic disease may be congenital as shown in the instance of a larynx from a child aged three and a half years, exhibited by Dr. Sturges at the Pathological Society. It is remarkable how much more common it is to find the chronic results of syphilitic disease in the air-passages, on examination of the dead, than acute affections, and yet as an accompaniment of a syphilitic laryngitis a bronchitis is by no means uncommon. To find an acute syphilitic ulceration of the bronchi is excessively rare, though we have a specimen showing the mucous membrane of the trachea and bronchi much ulcerated and flocculent, and which was believed to be syphilitic.

*Lepra.*—In leprosy the voice is usually hoarse, and laryngeal symptoms are amongst the most troublesome of the affection. Dr. Beaven Rake has sent to our Museum, from Trinidad, two specimens of the disease; the top of the larynx and epiglottis have undergone great thickening and the surface is ulcerated. Another example came from a man who acquired the disease in London, and whose models in wax are now seen in our Museum. In this case ulceration was found throughout the larynx and trachea.

In other cases which have been described the patients have died of chronic disease of the lungs; other descriptions of the morbid appearances in the larynx are not markedly different from those of tubercular disease. Dr. Thin found bacilli in a greatly thickened epiglottis from a patient who died of leprosy.

*Lupus.*—This occasionally attacks the larynx in common with other parts of the body. One specimen showing ulceration of the epiglottis was from a patient of Mr. Bryant, who regarded the disease as lupus. One other specimen, a more recent one, was supposed to be an instance of the same disease, both during the life of the patient and after death, the examination by the laryngoscope showing a thickening of the larynx of so peculiar a character that Dr. Pye-Smith believed it to be an example of this disease. After death by gangrenous disorganisation of the lung, the epiglottis was found quite destroyed, and the glottis with the parts around much swollen by thickening of the mucous membrane; this was raised, soft, and ulcerated. It approached more to the character of syphilis than any other ordinary disease.

*Xanthelasma.*—A specimen put up by Dr. Fagge shows yellow patches and transverse bands on the mucous membrane of the trachea. They resemble in appearance the atheromatous patches on arteries.

**A Cancerous Ulceration,** or destruction of the upper part of glottis and epiglottis, in connection with the same disease in the pharynx, sometimes happens, as may be seen in several preparations, though it is not very common.

**Typhoid Ulceration.**—One of the concomitants of typhoid disease is an affection of the larynx. It is not very common in this country, though it sometimes occurs, and several specimens of it may be seen in the Museum. It is generally described by Continental physicians under the name of *laryngitis typhosa.* At the posterior part of the larynx, at the junction of the vocal cords, we find a little hollow space on each side; this cavity is produced by the softening and sloughing of a deposit which occurs at this spot. In one case the cavity led into a space between the trachea and œsophagus, through which air had escaped, and had given rise to general emphysema. We may here remark, that emphysema has been occasionally observed in the course of typhoid fever, and in this affection probably may lie the explanation.

**Disease of the Cartilages.**—We have just now stated that very many of the chronic laryngeal affections have a syphilitic origin, and wherever the induration and thickening exists of which we have spoken, there is considerable suspicion of this being the case; there appears, however, to be an affection of the laryngeal cartilages independent of the specific taint, for in many instances there is no syphilitic history. The disease advances with considerable pain and swelling about the larynx, accompanied by suppuration either within or without. After the abscess is opened, the cartilage is seen to be dead and of a black colour, and like a piece of leather; very commonly, however, the portion of diseased structure, as one ala of the thyroid, is found converted into bone, which becomes exposed and exfoliates, if death do not previously ensue. The nature of the pathological affection is not altogether clear. When the inflammation has begun on the surface, the name *perichondritis* has been given, but, as in many cases of the disease an ossification of the cartilage has occurred, it has been thought that this is the commencement of the morbid condition, and that necrosis follows, attended by suppuration and exfoliation; whilst some think that during the slow inflammatory process, whether arising in the first place from syphilis, injury, or other cause, the change in the cartilage takes place. This affection of the cartilage is to be considered independently of the chronic laryngitis, where the mucous membrane is especially diseased, for it is accompanied by different symptoms, and runs a different course, although occasionally disease of the cartilage does appear to begin from within. Sometimes an abscess opens externally, and the cartilage is exposed; or the abscess may pass up and open into the pharynx, or the cricoid may be especially affected, and a fistulous opening leading from it to without. One of our specimens refers to the case of a child who died of laryngeal abscess with necrosis of the cricoid following measles.

We may here refer to several specimens, of which the pathology is very obscure, where an opening exists between the trachea and the œsophagus; in these cancer was suspected, but none was found after death; and, if due to simple suppuration, its cause was not evident. More rarely the hyoid bone becomes necrosed, giving rise to abscess and exfoliation.

**Ossification.**—This sometimes occurs in various parts of the air-passages. We have already said that in cases of disease of the cartilages, attended with suppuration, it is very frequently met with. In old persons the larynx is thus found ossified; also the rings of trachea and bronchi may show bony deposits forming plates and irregular patches all along the surface; these are quite independent of the rings, and are formed in the mucous membrane; the microscope showing true ossific structure.

**Adventitious Growths.**—These mostly occur at the upper part of the larynx, their favourite seat being on or near the vocal cords. The smallest and simplest of these growths are of a warty or *papillomatous* kind, as may be seen in several specimens on our shelves which came from children; one being on the epiglottis. When placed under water we see shaggy or villous processes float out, and they are then clearly seen to be attached to the vocal cord. They may sometimes grow to a large size until they block up the larynx or protrude into the pharynx. Sometimes these growths are softer and of a mucoid character, or they may be of a fibro-cellular nature, and then be called *polypi.* We have a specimen of a polypus which hung from the ventricle of the larynx and necessitated the operation of tracheotomy and the constant wearing of a cannula. One day the tube fell out, and death was almost instantaneous. Some years ago we had in the hospital a boy who had a *cyst* on the under surface of the epiglottis producing symptoms of suffocation. It was incised by Mr. Durham with complete success, a thick mucoid fluid escaping.

The *malignant tumours* grow from the same parts as the innocent ones, and are often primary. This is now known to be the case, through the use of the laryngoscope; an older opinion being that most of the cancers of the larynx proceeded from neighbouring parts. We find, however, that *sarcomatous*, true cancers and *epithelial* cancers spring from the vocal cords and adjacent tissues. Their surface is sometimes papillary, but very often rounded or lobulated. When the surface is papillary, it is very difficult to tell by the removal of a portion during life, what its true character is. It is the deeper portions only which may have capsulated or other cells distinctive of its real nature. It is remarkable how seldom the adjacent lymphatic glands are affected, and more especially in the sarcomatous variety. True *sarcomas* of round-celled and spindle-celled variety are met with as primary growths, also true reticulated spheroidal-celled growths of *carcinoma*, as well as the squamous-celled *epithelioma.* Our Museum contains growths removed during life by Sir A. Cooper and Mr. Cock. Cancer may affect the air-passages from without, as in cancer of the œsophagus, and occasionally may set up an inflammatory process causing external abscess.

**Injuries.**—Injuries from direct violence are rare, but those most usually seen are in cut throat. Mostly, however, the instrument of destruction passes between the cartilages, or rather above the thyroid between this and the hyoid, and so they are uninjured; if they should be incised, and union take place, it is, as we have before said, not by a reproduction of cartilage, but by fibrous tissue, which, in the course of time, may become bony.

Mr. Lane has found in the dissecting room numerous instances of fracture of the cartilages of the larynx and hyoid bone which had undergone complete repair, suggesting to him that these injuries are not so uncommon as is supposed.

A remarkable case occurred of injury to the windpipe, the specimen being now preserved in the Museum. The man from whom it was taken was crushed by a weight falling on his neck, which completely severed his trachea, the ends being two inches apart. He lived fifty hours after the accident.

**Foreign Bodies.**—This is a subject very important in practice, as the surgeon may often be called in to perform tracheotomy on account of extraneous substances having found an entrance into the windpipe. We have among our preparations coins and pieces of bone which have thus passed in. During life the substance may be found constantly moving up and down the trachea, endeavouring to make its exit, and first passing into one bronchus and then into another; when, however, it has become permanently fixed, and death has been the consequence, it has, we believe, been generally found in the *right* bronchus, or one of its branches, where it has produced a local inflammation, abscess, and destruction of the lung tissue. In the Museum is a specimen of an ear of corn which came out of an abscess in the back. It had been taken into the trachea and lungs, and ulcerated through the walls of the chest on the left side.

**Sputa.**—This is scarcely a subject for these lectures; but we would advise everyone to familiarise himself with the different appearances of the matters expectorated, as they are seen by the naked eye and by the microscope, such as the frothy mucus of bronchitis, especially where there is much difficulty in expectoration; the large quantities of uniform purulent matter in acute bronchitis; its nummular character in chronic disease of the tubes and in phthisis, and the rusty expectoration of pneumonia; also the black spit in some forms of chronic broncho-pneumonia, or the large quantities of pus (which is often fetid) from an empyema; then by the application of the microscope we find all these display, as a principal ingredient, granule- or pus-cells; the dark specks due to larger cells containing black points of pigment; the red expectoration showing blood-globules, and with these epithelial cells, and round nucleated pavement-cells from the smaller bronchial tubes or perhaps pulmonary vesicles; ciliated cylindrical epithelium from the larger tubes, and with these may be associated larger epithelial cells from the mouth. In cases of phthisis, where the lung is disorganised, fragments of the tissue may sometimes be found by placing portions of mucus under the microscope, or by taking a quantity of the secretion and boiling in an alkali. The solution is thrown into a vessel of water, and after standing the sediment is examined; the elastic tissue of the alveoli may thus be readily seen if destruction of the lung be going on. The tubercle bacilli may be seen by drying a little of the sputum on a cover-glass, and then testing it with a very dilute solution of caustic potash (Baumgarten's method). They may also be demonstrated by staining with fuchsin or magenta, according to the methods of Ehrlich, Weigert, or Heneage Gibbes, of which processes full details will be found in any Manual of Bacteriology.

## PLEURA

**Inflammation.**—Pleuritis presents various conditions, according as it is acute or chronic, and according to the kind of material effused. Although, as before said, acute diseases may pass into chronic, yet, very often, these affections are so different that they do not so much resemble each other as some diseases bearing distinct names; a fluid effusion, for example, on the one hand, and a thickened pleura, approaching almost in character to a new growth, on the other, have very little in common.

It is very possible that pleurisy may arise under two very different pathological conditions according as the pulmonary or the costal pleura is affected. In the former case it may be associated with pneumonia, or its cause may be coextensive with that which has produced the inflammation of the lungs; in the latter it may be of a purely local character. There may be, indeed, the same distinction as we draw between arachnitis having its cause from without in connection with the dura mater, and that which originates essentially from within in connection with the brain itself.

**An acute Pleurisy,** if seen a day or two after its first onset, is characterised by an effusion of lymph forming thin films over the surface of the pleura, and causing the lung to adhere to the chest. This thin layer can be easily peeled off the serous membrane, which is then seen to be of a dull hue, and at the same time highly vascular. The hyperæmic condition is best witnessed on the costal pleura, where the membrane is often intensely red; but on the lung, as it is frequently associated with pneumonia, the pleura presents a dull and whitish aspect. Even earlier conditions of inflammation are sometimes seen, where from a dull-looking pleura a very minute quantity of exudation may be scraped off; but generally much more effusion is seen than has been mentioned. Thus the whole lung may be covered with a layer of soft lymph completely hiding it, and the costal pleura be covered in like manner; and in such a case the lobes would be found adherent together, and the base of the lung to the diaphragm. We cannot say in what proportion these different parts are likely to be affected, but interlobular pleurisy, and that of the base of the lung, are probably as frequent as any. Combined with this effusion of lymph there may be some serum, which will collect in the meshes of the fibrinous exudation, and may be found in considerable quantity in the pleural space. Local patches of pleurisy may be found over the consolidated portions of lobular pneumonia, apoplexy of the lung, &c. After a moderate amount of lymph is thrown out, some absorption takes place, and the remainder forms into a fibrous tissue which causes the lung to adhere to the chest. If one of these adhesions be examined by the microscope it will be found that it is composed of fibres, the surface covered with pavement epithelium, and the black specks in it consisting of pigment-cells, as in the colouring matter of the lung.

Sometimes, and owing generally to a constitutional cause, the effusion becomes purulent, and an *empyema* is formed. The lymph which is thrown out forms a layer over the pleura both of chest and lung, like the wall of an abscess, and the matter is contained within. If the chest is filled, the lung necessarily becomes compressed, and, if not adherent, contracts close to the spine. It is doubtful whether this purulent matter ever becomes absorbed, and therefore it either leads to the death of the patient, or attempts to make its way out; unless, indeed, it be in small quantity, or circumscribed, when it possibly may remain inert for a considerable period. Not long ago, we had an opportunity of examining the body of a woman who had a local collection of pus, or an abscess, at the very bottom of the chest, between the lung, diaphragm, and ribs; this was shut in, and probably had existed for some years; for during this time there had been physical signs of fluid at the part, and a history of pleurisy three years before, and we think we have met with other similar instances. If the abscess in the pleura attempt to make its way out, it is generally either externally through the thoracic parietes, or internally through the lungs. In the first case, it is remarkable that the favourite site for its exit is not at the spot most favorable for tapping; that is, posteriorly, at the seventh or eighth rib, where we generally insert a trocar; but nature mostly evacuates the contents anteriorly and higher up. Thus we may observe a protrusion commencing as high as the third or fourth rib in front; after a time the abscess bursts, and the pus continues to discharge for many weeks through a fistula, during which time the lung may again expand, but generally the chest falls in, so that during the process of cure, considerable contraction of that side of the thorax is occurring, accompanied sometimes by a slight curvature of the spine. It is a rare occurrence for a localised empyema to make its way through the chest. If the matter make its way through the lung, it opens into a bronchial tube, is spat up, and is so got rid of; if the walls of the abscess should contract as the matter is diminished in quantity, no air need enter the chest; but sometimes as the fluid escapes, some air enters, and thus decomposition takes place, the purulent matter becoming very fetid. We have, then, in the chest, fluid mixed with air; in fact a *pyo-pneumothorax.*

In most cases the affection arises from the rupture of a diseased lung, and a pleuritic effusion is the consequence. In cases of empyema bursting into the lung the air escapes into the chest, which already contains fluid.

A cause of pleurisy, with more or less pneumonia, has been hitherto overlooked; viz. old disease and obstruction of the lymphatic glands.

On the surface of the lung the lymphatics may be seen taking their course full of pus, and also the lymphatics on the interlobular septa; so that a lymphangitis has been the primary affection.*

As *pneumothorax* is a distinct pathological condition, it requires a description of its own, but, as we are on the subject, we will complete our remarks upon it. Air in the chest arises either from its escape from the lung or its entrance through the thoracic parietes. It is remarkable, however, how rarely the affection occurs from injury to the walls of the chest, the opening being closed by elasticity of the skin, or adhesions of the lungs; and thus, as in the case of a police-officer who was shot through the chest, no air got into it at first; and it was only as the wound sloughed, and in the course of some days, that this occurred. In most cases, pneumothorax arises from escape of air from the lung into the chest, either from disease or injury; if the latter case, it is mostly from fracture of the ribs, and this is often associated with emphysema, or diffusion of air into the cellular tissue of the skin. Under these circumstances when the chest is opened, the lung is found contracted, and if death has been rapid there is no fluid except some blood from the lacerated lung; if death be not immediate, then some inflammatory effusions are present as well as air. Fracture of the ribs is not necessary to produce a laceration of the lung; for in the case of a child run over by a cart, the lung was torn, without any injury to the parietes; and another remarkable circumstance in connection with injury of the chest is, where after a penetrating wound, instead of pneumothorax taking place, the lung protrudes, constituting a hernia of the lung. We have seen several cases of pneumothorax arise from tracheotomy, and we mention the circumstance because we are not aware that it has ever been alluded to; in one case where, after tracheotomy, death occurred without sufficient reason, both the lungs were found contracted in the chest, and the cellular tissue in the posterior mediastinum was filled with air, producing large bubbles, which, we think, had burst through the pleura into the chest. In another case, where most extensive superficial emphysema followed the operation, the breathing became laborious before death, and the lungs were found contracted in the same manner; the emphysema having penetrated the mediastina. Most cases of pneumothorax arise from disease, especially phthisis; and this being one cause of sudden death, may have come on after the last visit to the patient; it is as well, therefore, always to percuss the body before opening it, and, if pneumothorax be suspected, a trocar should be thrust into the chest, when a gush of air escapes, which is audible, or will blow out the flame of a lamp placed near the opening. Most frequently, being not immediately fatal, a pleuritic effusion has occurred, and the case is one rather of hydro-pneumothorax, and thus, when the chest is opened, the lung will be found compressed, but not into a very small space, from its being diseased; the remainder of the chest, as the outer and lower part, will be found partly full of purulent fluid; if a tube be placed in the trachea, and blown into by the mouth, or by means of a bellows apparatus, the opening in the lung will be detected by the bubbles of air rising through the fluid; or, should the opening be above the level of the fluid, the air may be observed escaping; if not, the remainder of the chest may be filled with water, and the experiment be again made. On examining the lung, the opening will be found to be caused generally by the bursting of a small phthisical abscess in a very early period of pulmonary disease, and, this already communicating with a bronchial tube by a previous ulcerative process, the direct passage will be readily found between the main bronchus and the opening. The latter will generally be met with about the middle of the lung; and in the majority of the cases which we have examined it has been at the lower edge of the upper lobe; in one case, it was at the lower surface of the upper lobe. In speaking of the first form of this disease, where the primary affection is an empyema which makes its way into the lung, the cases are not sufficiently common on the post-mortem table to warrant a positive opinion as to how the opening of communication is formed, and what is its favourite site. In the phthisical form, the opening precedes effusion. As the empyema may be circumscribed, we apprehend it chooses the nearest spot of lung through which to penetrate; but in one fatal case we examined, after a most rigid examination, we failed to find any perforation, excepting, indeed, a number of small openings which were seen after removal of the lymph in the softened lung tissue. Such a case, may, perhaps, warrant an opinion which was held by Dr. Barlow, that the matter may be taken up by the surface of the lung, and discharged by the bronchial tubes, without any direct opening.

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* Moxon, 'Trans. Path. Soc.,' vol. xxiv.

It must be remembered that pneumothorax arising from an empyema breaking into the lung is not so common as that arising from rupture of the lung, due to septic or tubercular ulceration. In the former case a perforation of the lung may have occurred sufficient to allow of the evacuation of the matter, and yet no air escapes into the pleura in return. There may be a valve at the opening allowing a passage only one way, although the expellent force is itself sufficient to overcome the passage of air.

**Chronic Pleuritis.**—This may be essentially chronic, or result from an acute attack, and may be attended by various results, as effusion of fluid, serum, or pus, or a mere thickening of the serous membrane. So difficult is it to say in some cases where acute inflammation ends and chronic begins that we have spoken of empyema already, as the case where pus is poured out under acute affections, but very often it is essentially chronic. So, also, with effusions of serum; in some forms of pleurisy a very rapid pouring out of fluid takes place, not so quickly, indeed, as the more solid products of lymph, which occur in a few days, but generally in the course of about two or three weeks, when the chest may be found having some lymph lining it, but at the same time filled with a serous fluid. In cases where the exudation is wholly serous, the disease is essentially chronic, and is so slow in its progress that its presence would be unknown were it not for the physical signs. In these cases the chest is filled with pure serum, and the lung is compressed against the spine; generally the fluid is again absorbed, but if by chance an opportunity for examination should occur, the pleura presents no difference from a healthy membrane, and thus this *chronic pleuritic effusion* is like other serous exudations whose pathology is unknown, as, for example, hydrocele; we know, indeed, that the serous membrane is not in a healthy state, or the increase of secretion would not take place, but the deviation from the normal condition is seldom appreciable to the eye. If the lung has been compressed for some time, it becomes permanently carnified, and finally the whole tissue atrophies.

When the fluid is in small quantity the lung is not compressed as a whole, but only the lower portion corresponding in amount to that of the fluid secreted. The edge of the lung is found compressed and airless, whilst the remainder of the organ is uninterfered with.

As a chronic inflammation may result in effusion, so it may produce a mere *thickening of the pleura.* In one case this may have been preceded by an acute attack, and in another the increase of thickness is so slow in its progress that the change is more like a growth than a product of inflammation. In the former, the process from beginning to end may be understood on carefully examining the chest; thus, if death occur some weeks or months after the first attack, it may be found that the lung separates with tolerable ease from the thorax, the lymph on the surface being still soft; but on making a transverse section through the lung, the layer below will be seen more dense, and the new tissue below that still more so, until we arrive at that next the lung, which is the hardest of all, and incorporated with the pleura. The earlier layers are thus seen to be the hardest, and the recent ones the softest; this we call a *chronic* process: not one synonymous with *old*, or one that is altogether past, but one which commenced at some antecedent period, and continued up to the time of examination. Another chronic process, but still slower, is where there is no evidence that at any time a soft material has been effused, the increase of thickness being imperceptible, and more allied to a growth; it is a question with some, whether such a thickening *must* not have been preceded by a softer effusion of lymph; but from the observation of several cases where there has been no history of any acute action having taken place, we are quite of the opinion that this is not necessary, and that such thickened pleura need never have presented any other appearance as regards its texture than that actually found at the termination of the case. In these instances we see the lung closely adherent to the chest by tough tissue as hard as cartilage, so that the knife is required to cut it out; in one man, we remember, this new tissue was an inch in thickness, and in the case of a boy one inch and a half by measurement, and was associated, as is usually the case in these severe forms, with cirrhosis of the lung; the whole organ being sometimes affected, at other times merely a part, and the more frequent place the upper lobe. The question here alluded to, of the mode of formation of such a tissue, is one still open and discussed by pathologists; the point being whether a preliminary stage of effused lymph is necessary for its production, or whether the process is not rather allied to a slow growth; that is, whether an exudation, which is at first separate, subsequently becomes organised and attached to the original surface; or whether the new tissue has ever been separate from the latter, or has rather been an offshoot from it. We think that both occur, and that one does not preclude the other; it is true, no doubt, that an effused lymph may subsequently become organised and form a part of the original tissue from which it proceeded, but, at the same time, that the other is none the less true; that is, that the increase to the surface is so slow and to so slight an extent, that no actual separation from the original membrane could ever have been witnessed; it may commence, however, in the form of elementary cells, as in a positive exudation, for even in well-formed new growths of a fibrous character, which go by the name of tumours, the nucleated fibres are preceded in most cases by a production of such cells. In some very chronic cases of phthisis attended by a thickened pleura it is a question whether the disease has not been primarily pleuritic, and the fibroid process proceeded inwards.

**Morbid Growths.**—We have already said that in cases of cirrhosis of the lung, where the pleura is excessively thickened and indurated, the process is allied to a growth. Sometimes, in such cases, an earthy deposit takes place, or an ossification, as it is called, in the new tissue, and a large plate of bone the size of the hand may be found on the surface of the lung, or in some cases at the base of the lung, where the latter adheres to the diaphragm. Such large bony pieces may be seen in our specimens. Such plates are called ossifications, as also the thickening of the pleura styled cartilaginous; but it must be remembered that all that is meant by such expressions as cartilage-like, and bone-like, is, that in the one case the tissue is simply fibrous, and in the other consisting of a matrix of simple fibre containing earthy matter or salts of lime contained in the meshes of the inflammatory product.

*Bone* and *cartilage* may, however, occur in the lung, and therefore on the pleura, but, if so, generally as deposits secondary to similar growths in the external parts of the body, and very rarely as primary; so rarely, that in the one or two cases where we have seen them, it is a question whether, if the whole body had been carefully examined, some similar growths might not have been found elsewhere. In a case not long ago examined we found small bony plates on the pulmonary pleura, and these, when viewed by the microscope, were found to be composed of true osseous tissue. There is in the Museum a mass found in the pleural cavity, and said to be fibro-cartilage, but we have not examined it.

*Fat.*—A few instances have been recorded where a layer of fat has been found covering the lung.

*Cancer.*—When cancer involves the lung it may also attack the pleura, and it generally occurs as a secondary deposit; in these cases it is scattered all over the pleura, and sometimes without the lung itself being involved. It is generally seen in the form of white, flat, and smooth patches, scattered all over the pulmonary surface, but penetrating occasionally into the lung tissue. These are generally very hard, being composed of fibrous structure as well as of epithelioid cells, and if only in small quantity, are not easily recognised, nor the idea of cancer suggested, unless this is seen also to prevail in the abdomen, or elsewhere. The costal pleura may be similarly affected, and sometimes severely, by large vascular medullary growths springing from its surface, or occasionally by cancer actually growing through the thoracic walls, from the exterior, as in carcinoma of the breast. Dr. Pitt had a case where cancer was found in the pleura and other parts. He believed that the growth in the pleura was primary.

*Tubercle.*—In cases of general tuberculous infiltration of the lungs the pleura is frequently similarly affected, and thus the whole surface may be found covered with small miliary tubercles. There is also another form of more acute deposition, which might also be called tubercular pleuritis, where in the case of an inflammation an effusion takes place in a subacute degree, but of a tuberculous character. For instance, a boy, after receiving a blow on the chest, had a pleurisy set up; and at the time of death, three or four weeks afterwards, the lung, when torn from the thorax, was found to have a yellow tubercular matter effused upon it, or a lymph putting on the tuberculous character.

Amongst the morbid contents of the chest we have mentioned *air*, *serum*, and *blood*, and these have already been spoken of under several heads. We have said *air* may get in from without through the chest, in case of an opening in the parietes, but more usually it enters by or from the lung, and this occurs either from injury or disease; in the first place, mostly from a fractured rib lacerating the organ, or its being punctured by the stab of a weapon or bullet. It is remarkable that the latter does not always cause pneumothorax, even if the organ be considerably injured. If from disease, it is generally from rupture of the lung occurring in phthisis. Dr. Hughes relates a case where he thinks air escaped from an emphysematous bubble; but, as recovery took place, there was no opportunity of verifying the diagnosis.

*Serum.*—We have already alluded to this as one of the products of inflammation, but it may also be a passive effusion. It is thus found associated with dropsy in other parts of the body, in connection with heart or kidney disease. It is often difficult, however, to know why it should occur on one side rather than the other; but in Bright's disease of the kidney we may often find a very simple explanation in the fact of one lung being adherent, and, there being no space for the effusion, the lung itself being œdematous; while the effusion occurs on the other side where there are no adhesions, and the lung is consequently compressed. It may arise altogether from a local cause, as when pressure occurs on the pulmonary vessels, as in cancer of the bronchial glands, and thus is a very common termination to cases of carcinoma mammæ. This passive effusion is called *hydrothorax.*

*Blood.*—Blood in the pleural cavity may have many sources; thus, a rupture of an aneurysm may cause it, or a wound of the lung from fractured ribs. In such a case, a short time ago, where death occurred after some days from pleurisy, lymph was found mixed with blood, forming a layer on the surface. It may also, occasionally, arise, as we have a few times seen, from laceration of the liver, the lower ribs being fractured. The diaphragm and liver have been torn, and blood has been effused into the chest. This, of course, is when the right side is injured; a similar injury on the left side, with laceration of diaphragm, would tend to diaphragmatic hernia, or displacement of the stomach into the chest. In all such cases the source of blood is manifest; but there is one case where blood is simply mixed with inflammatory exudation, as in *hœmorrhagic effusion.* We have already alluded to a similar state in the pericardium; it arises probably from some constitutional cause, where there is a tendency to purpura, and thus, after the pleurisy has existed for some days, blood is poured out. On opening the chest, the lungs seem compressed, and the cavity filled apparently with blood; but on careful examination the serous membrane will be seen to be covered with lymph, and in its meshes blood is effused. Whether the source of this is the original tissue softened by the inflammation, or the rupture of new-formed vessels, has not yet been satisfactorily determined. In cases of *injury to the chest,* from stabbing or gunshot wounds, death often ensues from a pleurisy in which inflammatory products are found mixed with decomposed blood, the source of blood having been either a wounded intercostal vessel or wounded lung.

.*Contents of the stomach* may sometimes be found in the chest; it is not common, but we have seen it two or three times. It is known that the stomach becomes dissolved sometimes by its own juice, and the contents escape into the abdomen; in the same way, from the body lying on its back, some of the gastric juice passes into the œsophagus, and, there causing a similar solution, the contents may escape into the chest, and then, indeed, begin to act on the lung, softening and dissolving its structure. It is very important, sometimes, to recognise such post-mortem changes, and distinguish them from ulcerations; for it was in a case of poisoning by arsenic that one of the cases of digestive solution of the œsophagus occurred.

We have already alluded to the case of diaphragmatic hernia, where the stomach, or transverse colon, may pass through a ruptured diaphragm into the chest.

## LUNGS

**Hypertrophy.**—We know very little of hypertrophy of the parenchymatous organs, as we shall presently have to say with reference to the abdominal viscera; that is, in an absolute sense, although a relative hypertrophy is common enough, the wasting of one part (or of one organ when it is double) being compensated for by the enlargement of another; and thus, as regards the lungs, when one of them has been long atrophied the other becomes larger, and thus it is seen not only filling its own side, but crossing the median line and heart and reaching to the opposite side. We would not, however, positively assert in such a case that the lung is actually hypertrophied, because enlarged; but, judging from the analogous case of the kidney, we see no objection to that view.

**Atrophy** arises from various causes. The most simple case is where the lung has been long compressed from pleuritic effusion or thickening, when the tissue has eventually undergone a structural change,—in fact, become atrophied. The same may occur in a part of the lung, as presently to be mentioned, or a lobe may be so affected by chronic pneumonia as to waste away.

**Condensation of the Lung—Atelectasis, Apneumatosis, &c.**—This is the condition of a lung while in the fœtal state, or while in utero, which has never yet contained air. The cells are collapsed, and thus it constitutes a solid mass, which sinks in water, like any other organ. After the lung has been used, if the air be squeezed out the same state is again produced; this is well seen in pleuritic effusion, where the lung becomes compressed against the spine, and when taken out is quite airless, and sinks in water; this is at once distinguished from a hepatized lung, which also sinks in water, by being tough and resembling a fleshy substance; hence the name *carnification.* The air in this case is forced out by external pressure, but the same thing may occur in parts of the lung from mere want of power in the respiratory process to overcome the natural elasticity of the tissue, and, consequently, certain lobules, or larger portions of the lung, may return to the fœtal state, and, inasmuch as this resembles the same condition where, from debility of the child, the lung has never been thoroughly filled, the name *atelectasis,* signifying imperfect expansion, has been given to it. This is congenital atelectasis.

In infants who survive their birth but a few days, the lungs may be thus found imperfectly expanded, portions being quite airless, dense, and sinking in water. At a later period of life, the same state may still be found, and then it is very probable that the lung may have once been wholly expanded, but, owing to some weakness of the patient, portions have again resumed their original fœtal condition. If, indeed, this does not occur from mere inability of inflation, it has of late been very satisfactorily proved to arise from any cause which may obstruct the entrance of air into the lung. We believe the first person who directed attention to this return to the fœtal state in certain portions of the lung was Dr. Alderson, who, several years ago, pointed out how this condition is often found in conjunction with hooping-cough, and that it had hitherto been looked upon as lobular pneumonia. Dr. Bright also had observed the same fact.* In speaking of hooping-cough it might be observed that this, of late, has been regarded by some as a parasitic disease, and that a micrococcus has been found which irritates the mucous membrane of the larynx. Collapse of the lung may, however, follow ordinary bronchitis, especially in children. The lung, which in a child is of a light colour, is seen to have a number of dark red portions on its surface, and towards the periphery wedge-shaped masses; these were formerly supposed to be hepatized parts; they are more solid than the healthy tissue, and often sink when placed in water, but, unlike a hepatized lung, the surface is not granular when a section is made, and, moreover, these parts are depressed below the surface, whereas a mass of inflamed lobules would be raised. This is, of course, what might be expected when in one case the cells are filled with an albuminous matter, and in the other they are simply collapsed or airless; they are both increased in specific gravity, but one is enlarged, granular, soft, and exudes a fluid on pressure, while the other is diminished, smooth, tough, and dry. The proof we have of this being its true condition is in the power we have of artificially inflating these portions, and thus restoring the lung to its original state. We should say, however, that the two conditions may be combined; that is, an inflammatory exudation may occur in the collapsed part, for when the collapse occurs from the plugging of a bronchiole an extreme congestion naturally occurs. The collapsed portion is red, subsequently may become œdematous, and, finally, may be the seat of inflammatory changes. It is a state especially met with in children, but also in old people, particularly in the bronchitic, where a collapse of a large portion of the lung may occur, especially at the posterior part, and accompanied by recent inflammatory effusion; thus, portions of the organ are soft, easily broken down, and exuding serum, while others appear simply airless, but are firm and dry. A more than usual interest is attached to this subject, since it has been made the foundation for a new theory of emphysema, as we shall presently mention.

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* Dr. Bright, in his 'Medical Reports,' published in 1828, has the following:—"I had an opportunity some time ago of examining the chests of two children who died of hooping-cough. In both the breathing had become more and more laborious until death. There was not a vestige of what we would ascribe to pneumonic inflammation, but the greater part of the lung was in a state of decided emphysema, while many of the lobules about the edges of the lungs were flattened as if they had not admitted the ingress of air for a considerable time. The bronchial tubes were a good deal loaded with viscid matter."

**Emphysema** is divided into two forms, the *vesicular* and the *interstitial.* The first is the more common, and that which is generally implied when the term emphysema is used, although it so happens that to this very case the term is not strictly applicable, for, as used elsewhere, it means an infiltration of air into the tissue, which, in the case of the lung, is a comparatively rare occurrence.

*Interstitial* or *interlobular emphysema* is the more rare form, and is due to the escape of air beneath the pleura and into the tissue. It is generably recognisable by a number of air-bubbles beneath the pulmonic serous membrane, appearing like a number of beads scattered on the surface, and it mostly occurs on the anterior edge of the upper lobe. It is commonly found in the lungs of children who have long been subject to bronchial affection, as hooping-cough, and in these we see the surface of the lobe covered by a number of air-bubbles. We have also met with it several times as a consequence of tracheotomy.

The *vesicular* or *ordinary emphysema* is an affection which has of late attracted much attention, from the novel theories respecting its mode of formation, and therefore we will venture upon a few particulars concerning it. Emphysema, when existing to any extent, is at once recognised, and is constantly seen in old bronchitic cases, in the post-mortem room. The lung appears to have a number of additional pieces or bladders attached to its surface, and some of them are pedunculated. They are seldom observed except along the front edge of the lung. When cut through, they are seen to be composed of a highly rarefied tissue, like sponge, or the lungs of a reptile, consisting of large cells or spaces, which are either immensely dilated pulmonary vesicles, or several vesicles which have run together from the rupture of their partitions. It is mostly in old cases of bronchitis that this condition is met with, but not uncommonly also in chronic phthisis. The connection of emphysema and these diseases has always been recognised, and as consequent upon them. Bronchitis is evidently a very usual cause, and thus Laennec, who was almost the first to study diseases of the lungs by a philosophic method, framed an explanation which has generally been received up to the present day. He thought a bronchial tube became obstructed by mucus, and consequently, the air in the pulmonary tissue leading from it could not escape during the expiratory process, and therefore, from the great compressing force upon this part of the lung, the air distended the vesicles or burst through the tissue. Although such a process could not be absolutely proved, and there were many difficulties in the way of explanation, yet the evident connection of emphysema with bronchitis, and the many reasons to believe that it occurred during violent fits of coughing—that is, in the expiratory process—caused most physicians to follow Laennec, and, consequently, his doctrines were for many years taught in this theatre, even in spite of the promulgation of the more recent theories. The latter theories have resulted from the discoveries made with respect to collapse of the lung, or atelectasis, as we just now explained, and for their full development we are mainly indebted to Dr. Gairdner. It is said, in the first place, in opposition to Laennec, that any obstruction of a bronchial tube could not cause the retention of air behind it, but rather the opposite condition, for any plug of mucus in a conical tube would act as a valve, and thus, although not admitting air, it would easily allow the egress of that already in the vesicles; and to prove this, experiments have been instituted on the lower animals, in which bullets were placed in the bronchial tubes, when the tissue to which these led, so far from becoming expanded with air, became collapsed; the ball, acting as a valve, allowing air to escape, but not to enter. This immediately refuted Laennec's theory, for, so far from emphysema being produced, the lung contracted and became dense, in the manner already mentioned. Another objection, too, given is, that a pressure on a part of the lung during the expiratory process could not cause it to dilate, for equable pressure on a membrane full of air, however delicate, cannot alter its form; again, if the disease were produced after Laennec's idea, those parts of the lung should be found emphysematous where the tubes are most liable to obstruction; but the opposite is the case, the one occurring at the anterior parts, and the other at the posterior. With these objections, Dr. Gairdner has expressed his opinion that emphysema occurs not during expiration, but during inspiration; and that, although the first step in the process is in most cases a bronchitis, this is only effectual in producing a collapse of a portion of the lung, which is the condition immediately antecedent to the dilatation of another part of the tissue; the latter, indeed, being compensatory to the previous contraction. He thinks a tube, and this is generally one leading to the posterior part of the lung, becomes obstructed; that the portion of lung to which it leads collapses; and that, consequently, during inspiration, other portions, generally the anterior, become distended, to compensate for the proportional decrease; that the chest, indeed, when it expands, must be filled by a certain bulk of lung, and that, consequently, when one part of the tissue has become contracted another distends. Dr. Gairdner does not say, as some have misunderstood him, that when a part of the lung is functionally obstructed, as in pneumonia, another part expands to make up for its loss, but simply that, when one part is contracted, another expands to supplement its bulk in the chest. Again, if the tissue of the lung should contract from any other cause besides bronchitis, emphysema would occur, and thus its existence in very chronic cases of phthisis, where portions of the tissue are found puckered up by tubercular or inflammatory deposit. This theory was readily received by many, although, as before said, several of our older and most experienced physicians held by Laennec; and declared that, although there might still be difficulties in the old views, yet they could not but think that emphysema occurred during expiration. Subsequently Sir W. Jenner advocated this view, as opposed to Gairdner's, and maintained that it is during the expiratory process that the disease really occurs. He does not believe, with Laennec, that it is due to a compression of the air behind an obstructed bronchial tube, for the objections already stated are, no doubt, insuperable; but he considers Gairdner's simile of equable pressure on a bladder of air not true of the lungs, for the pressure of the chest is not equal; the lower portions of the lungs may have an equable pressure exerted on them by the posterior part of the chest together with the lateral part and floor, but the anterior part of these organs towards the mediastinum and the apices beneath the clavicles have not the same force exerted on them, and consequently if the whole lung were more than usually pressed by the thorax, these latter portions would endeavour to protrude and become distended. The expiratory force, therefore, is quite equal to the production of emphysema in those parts where the pressure is less, and in cases of violent cough, the apex of the lung may be actually seen forced up above the clavicle; moreover, this condition may occur without any appearance of compensatory contraction in any other part. In the case of M. Groux, who had the fissured sternum, the anterior part of the lung bulged through the opening during the act of coughing. And if the example of draught animals be taken, we find emphysema occurring merely from the strain upon the lung, and without any previous bronchitis or disease of the tissues; the strain being during the expiratory process, when the chest is expanded and glottis closed, so as to make a purchase for the muscles.

It will be seen that one of these theories supposes that the distension of the air-cells takes place during inspiration, and the other that it occurs during expiration. The first is also called compensatory or complementary, and is seen in cases when the lung has undergone contraction in parts as in chronic phthisis. The second is observable more frequently in cases of long-standing bronchitis. These theories are therefore not incompatible, for both are probably true. They do little more, however, than account for expansion of portions of the lung, and both are dependent upon a purely mechanical causation; but, after all, the most important form of emphysema is that when the whole of both lungs is affected, and where there is often no history of a previous bronchitis or cough. The case to which we allude is that where the chest has undergone a change in shape, where from the raising of the shoulders or the falling of the diaphragm it has become rounded or barrel shaped. In this case the lungs are of large size, are found after death filling the chest, feeling doughy to the touch, and when closely examined show the distended or ruptured air-vesicles plainly visible to the naked eye. The whole lung may be found affected in this manner, and at the same time there is no appearance of contraction of any portion, nor need there be any history of bronchitis previous to the time at which the symptoms of emphysema first exhibited themselves. We are forced, therefore, to the conclusion that the cause of this universal structural change must have been inherent in the tissue of the lung itself. When examined it is found wasted and the blood-vessels diminished in number and size; the alveolar walls may be found to contain fatty granules. When just now we said that this worst form of emphysema had not a purely mechanical origin, we would not deny that the inspiratory efforts when acting on a weakened tissue might directly produce the distension of the air-vesicles. Why the pulmonary membrane should have become weakened is not always obvious; but in some cases we meet with fibroid changes in the lungs as if an interstitial pneumonic process had been present, and under these circumstances we can see how a stretching of the tissue might have been produced. Villemin states that a fatty degeneration occurs in the nuclei found on the alveolar walls, that these give way and produce openings between the alveoli. Subsequently these rents enlarge until large hollow spaces are formed.

That a primary wasting of the tissue will cause the breaking down of the alveolar walls, and so give rise to emphysema, is evident from its common occurrence in old age, when the whole lung is atrophied, and at the same time emphysematous. In such cases the whole thorax becomes diminished in size, and in this way are produced the stooping posture and sloping shoulders so characteristic of old age. This senile change is often called *atrophous emphysema*, in order to distinguish it from the *hypertrophous emphysema*, where the chest is large and rounded, but the term hypertrophy must be here understood to imply no more than unusually large. In these cases the bronchial tubes are often found full of secretion, and their walls thickened, with great hypertrophy of the longitudinal muscular fibres.

**Hyperæmia and Congestion.**—A congestion of the lung is constantly met with in cases where the blood is delayed in its passage; it is seen often in perfection in cases of paralysis of the chest where the spine is fractured, the lungs being exceedingly gorged, and sometimes to such an extent that the blood oozes through the bronchial membrane, and is extravasated into the tissue. In very many cases of bodies we examine we find the posterior part of the lung much gorged with blood, and of a dark colour; this is due to the supine position of the patient some time before death and probably to the commencing softening of the tissues. This is known as *hypostasis*, and passes into an inflammatory state, presently to be mentioned.

A spotted condition of the lung, even if due to extravasation, does not necessarily show a mechanical impediment, for it may be due to a diseased condition of the blood or the tissues, and be of a *purpuric* character; thus, in true purpura, the lung as well as other parts of the body will be found spotted, both on the surface and the interior, also in many other organic diseases, where the same tendency to purpura exists, *e. g.* enterica, as well as in cases of manifest blood-poisoning, as pyæmia, &c.

A very long-continued congestion, as seen in heart disease, gives rise to a very peculiar condition, which is much in want of a good name to characterise it, for it is not mentioned by some authors, and by others classed with pneumonia. The name *splenization* having been first given to this condition, although since applied to a stage of inflammation, has generally been adopted; it may be remembered that, as a consequence of the long-continued congestion in heart disease, various necessary changes occur on the serous and mucous surfaces, and in the parenchymatous organs a remarkable hardening takes place, noticeable especially in the kidneys and spleen: in like manner the lungs are indurated, the organ feels bulky and heavy on removal, suggesting a hepatized tissue, but, on section and more careful examination, the structure is found dense and tough, and containing but little air. When scraped it is fleshy, giving out no secretion, and instead of being granular the surface is glazed and shining. In parts of it, blood may be found actually extravasated, constituting apoplexy; but, without this, you may sometimes find a whole lobe solid, almost airless, and at the same time quite tough when an attempt is made to tear it. There are various degrees of this: when slight, the lung is red, and resembles a hardened spleen, and in this case a microscopic examination shows little else than stagnation of blood in some of the smaller vessels, including also the lymphatics; subsequently the vessels become distended and tortuous, and thus encroach on the air-cells. In more extreme cases this redness is lost, and the lung assumes a browner shade; and when in this condition the microscope shows abundance of corpuscles and granule masses, a chronic inflammatory state is no doubt present.

When in the simple mechanically congested lung a quantity of pigment is formed, and the alveoli are thickened, and the lung tissue is dark and granular, the term *brown induration* has been applied; in these cases the capillaries have sometimes become varicose. If beyond this condition inflammatory products are thrown out, some formation of connective tissue takes place, and this consolidation, combined with the granular pigmented condition, has given rise to the name *brown indurated pneumonia.* The accidental introduction of blood into the lungs may set up a pneumonia, and consequently it is not uncommon to see a fatal inflammation of the lungs follow a hæmoptysis. The blood passing into certain lobules there sets up an irritation and a pneumonia results. There is generally a high degree of fever, associated with the physical signs of consolidated lung, and the expectoration of blood and gelatinous sputum. After death the lung will be found to be the subject of lobular pneumonia mixed with extravasated blood. If the case be not immediately fatal, it is believed by some that the masses of lobular pneumonia undergo caseation and soften; and so a phthisical condition is produced. It should be mentioned that there are good authorities who are not ready to admit the fact of a pneumonia being set up by the presence of blood, much less that it may pass into a chronic process and be the origin of phthisis, the so-called *phthisis ab hæmoptoë.*

Sometimes in persons who have been suffering from disease of the brain, which interferes with the respiratory process, the lung may be found remarkably tough, fleshy, and firm. A remarkable instance of this occurred in a case of paralysis of the diaphragm due to a nervous cause.

**Apoplexy.**—If the pressure on the blood-vessels be very great, the blood is actually effused into the tissue, and we have what is called *apoplexy* of the lung—a very absurd name, but one now in general employment. The blood gradually oozes out, and infiltrates itself into the tissue, both into and between the cells, and there coagulates and hardens; so that it does not necessarily happen that any of the fluid escapes into the bronchial tubes, or that there is any hæmoptysis during life. Owing to this gradual exudation and coagulation of blood the part thus affected becomes hardened, and the circumference is quite defined from the adjacent healthy tissue. The apoplectic condition is thus recognised on the surface of the lung before it is cut into; the part is enlarged, as it would be if hepatized, but instead of the induration passing off gradually into the adjacent tissue, it is defined by a distinct boundary, and thus it is felt like a tumour in the lung. A section at once shows its nature, and distinguishes it from hepatization; it is seen to be blood, and of a black colour, and resembles (to use the common simile) damson cheese, the texture of the lung being scarcely discernible, the branch of the pulmonary artery leading to the part being always plugged with fibrinous coagula. There are, however, cases where inflammatory products have been poured out, and thus a hepatized and apoplectic condition may be often combined; the section of an indurated lobe being white and granular in parts, and having small masses of blood in others; moreover, on the pleural surface it is not unusual to find a thin layer of lymph, showing the inflammatory process which has been present. Besides this, however, we may often find white fibrinous masses in the lung, indicating older effusions, the hæmatin having become decomposed and absorbed, leaving the more solid part of the blood as a white mass; and as this contracts, so does often the lung tissue with it; and thus is produced a puckered condition of its edges, with sufficient traces of the apoplectic state about it to show its pathology. In Addison's paper on pulmonary disease in the 'Reports' is a description and drawing of a case of a hard red fibrinous mass attached by slender adhesions to a closed cavity around it. In old cases of mitral disease these various changes in the apoplectic masses may be seen, but not often the puckered state showing complete absorption, which probably is to be referred to some previous temporary attack of obstruction in the circulation. The favourite seat for apoplexy is the circumference of the lung, and especially the edges, as the lower edge of the upper lobe and upper edge of lower lobe. This is probably owing to the circulation being less free. A portion of tissue in the middle of the lung would have anastomosis all round it; also the right lung appears to be more usually affected than the left; the explanation of which is not readily apparent.

Many of these well-known appearances of the lung in connection with heart disease have been shown of late years to have a different pathology; that these hardened and wedge-shaped masses are due to infarction or actual blocking of the pulmonary arteries by portions of fibrin carried from the heart; that, just as infarction or embolic masses are formed in the spleen and kidney in connection with endocarditis, so in like manner they may take place in the lung; that vegetations from the valves may be actually carried to a branch of the pulmonary artery, and there stick fast, or that smaller embolic particles washed off by the current of blood may give rise to coagulations in the arterioles, and so bring about the appearance known as an infarction. Probably the condition of lung above described, where the mass becomes developed and hardened, has had an origin of this kind. When in connection with endocarditis as of the mitral valve, infarctions are found in the spleen and kidney, which we call embolic, we cannot hesitate to give the same name to exactly similar appearances found in the lung.

In obstructive disease of the heart the infarctions are thought by some to take place in like manner; that a coagulation takes place in the right side of the heart, and portions of fibrin are carried into the vessels and block them. These emboli are innocent and must be distinguished from many others which are infective in their nature, and set up inflammation and abscess.

It is remarkable that this apoplectic condition does not follow the congestion of bronchitis, but only that of heart disease; this is due probably to the seat of stagnation being different in the two cases: in the one, the venous blood, though traversing the capillaries with difficulty, passes on, whereas in the case of heart disease the blood is detained from mechanical causes in the pulmonary vein, whence it escapes into the tissue; the blood therefore, in apoplexy of the lungs, would be from the pulmonary veins rather than the arteries.

There is also another condition, which is sometimes called apoplexy, arising from a different cause, and producing a *spotted* condition of the lung, whereas that just described forms *circumscribed* masses. It arises from the entrance of blood into the air-passages, and its diffusion into the cells. The fluid passing down a tube, and entering one branch rather than another, small portions or lobules of the lung become infiltrated, producing this spotted appearance. It is met with in those who have died from hæmoptysis, and especially in cases where the lung has been seriously injured by a fractured rib, leading to death by the drawing of blood into the tissue.

**Œdema of the Lung.**—This is a condition very frequently found in Bright's disease, or where there is a disposition to dropsy; the lung is found filling the chest, and heavy, and on a section being made a quantity of serum drains out, leaving the tissue healthy and firm. It is thus distinguished from the first stage as inflammation, in which the texture is very lacerable. Sometimes, however, when a person has been long dying, the texture may soften, and then if, as in renal disease, we know the lung is equally inclined to be œdematous or to be inflamed, it is difficult to say whether the serous infiltration be active or passive. This difficulty has given rise to the term *inflammatory œdema.* In some cases, the lung is found œdematous, whilst the tissue is dense and airless.

**Pneumonia.**—Although we describe pneumonia only in its anatomical characters it may be as well to remember that its causes and varieties depend, in all probability, on constitutional conditions. Cold and wet are spoken of as its more immediate antecedents, and it is true that the disease occurs more in inclement weather, yet it is clearly very frequently but the outward manifestation of some deeper or more hidden cause. Thus, in epidemics of influenza, some deleterious atmospheric action rapidly carries off the young and healthy through acute inflammation of the organs of the chest. It often occurs also during the progress of many chronic diseases, and it is frequently the termination of many debilitating states. Pneumonia may also at times be endemic, and is occasionally seen to pass through a household. All these circumstances point to some morbific causes instrumental in its production. The discovery by Friedländer of the *pneumococcus* may throw some light upon its causes and infective characters. Pneumonia is generally divided into simple or *lobar* pneumonia, where the whole lung or a lobe is affected, and into *lobular* pneumonia, where distinct and scattered lobules are inflamed. We also speak of a *chronic* inflammation, where the connective tissue between the lobules is supposed to be primarily affected leading to a general solidification and induration of the pulmonary tissue, under the names of fibroid changes, cirrhosis, and analogous conditions. The lobular pneumonia is usually associated with a bronchitis, and is mostly met with in children, or under some particular circumstances as during measles. Of late years, and more especially since the microscope has been put in requisition, it has been maintained that this lobular or broncho-pneumonia is not a modification in form or degree of the ordinary pneumonia, but differs from it in kind. It must be remembered that the mucous membranes are said to undergo two kinds of inflammation, both of which may be observed on the air-passages,—the one where the secretion is corpuscular or muco-purulent, and the other where the secretion is fibrinous or croupous;

as is seen in ordinary croup, where the larynx and trachea are covered with a membrane. In the one case the microscope shows a mass of cells formed by the proliferation of epithelium; whilst, in the other, the membrane is composed of fibrillated lymph containing cells in its meshes. Now, it is maintained that the inflammatory products poured out into the air-vesicles in ordinary pneumonia are of this latter character, and thus of late years many writers have given to it the name of *croupous pneumonia;* whilst it is also said that the lobular pneumonia partakes of the character of the bronchitis with which it is usually associated, that it is corpuscular in its formation, and would with more propriety be styled *catarrhal pneumonia.* It must be understood, then, that the term croupous pneumonia is applied by modern writers to ordinary pneumonia; whilst the term catarrhal is made to apply to what has hitherto been styled lobular pneumonia, or more correctly broncho-pneumonia.

*Simple or croupous pneumonia.*—It was long taught that inflammation of the lungs consists in the exudation of a lymph into the interstitial substance or parenchyma of the lungs, but of late years the doctrine which was always taught by Addison in this school, who was the first to demonstrate the true nature of pneumonia, is now universally adopted. It is the air-vesicles or the alveoli constituting the dilated termination of the bronchi which contain the inflammatory products. Addison was led to the conclusion fifty years ago by actual observations as to the seat of the exudation, by the structure of the pulmonary cells, and by the proved absence of any supposed intercellular spaces into which lymph could be effused. He observed that the changes which take place in the lungs are such as are seen in serous rather than in mucous membranes.

These changes are of the same kind as are witnessed in other structures, viz. first, an intense hyperæmia followed by a fluid and solid exudation. The alteration, therefore, which the organ undergoes is a most striking one, being none other than the conversion of a light spongy organ normally full of air into a heavy solid mass, and all within the space of a few hours. The different stages of the process are sufficiently well marked, and are traceable during life by various physical signs which accord exactly with the condition of the lung. They resemble what is seen in other structures during inflammation, viz. hyperæmia and vascular engorgement followed by an exudation.

The first marked change observable in the lung after death is that of *engorgement with serous exudation*, and this is usually called the first stage of pneumonia. On taking the organ out of the chest it is found to be more bulky and heavy than the healthy lung; it pits on pressure, and when cut through it is much redder from sanguineous engorgement, and a quantity of serum pours from it. Owing to the lung still containing air the serum is frothy, and portions of tissue still float when cut off and thrown into water. If now the finger be forced into the lung it will be found to be readily lacerable, and this is a means of distinguishing it from simple œdema or dropsy of the lung. This softening is one of the best signs of inflammation, and it further shows that the fibrous tissue which forms the walls of the pulmonary air-vesicles is more or less involved in the inflammatory change; for although we give up the old theory of an interstitial inflammation, yet we must hold that in connection with the cell-formation which takes place the texture of the lung is loosened by an exudation into its meshes, in the same way as in inflammation of other structures; as, for example, of the peritoneum, where it may be observed that, although the exudation takes place from the surface of the serous membrane, yet the coats of the intestine are infiltrated with lymph, and are readily separable from one another. Those who fail to find any exudation in the meshes of the alveoli would account for the easy laceration of the lung by the complete filling of the alveoli whereby they would offer resistance and give way on pressure. It is during this stage of serous exudation in the lung that fine crepitation is heard by the ear.

It is not usually until the lung has reached this stage in the inflammatory process that inflammation is recognisable during life, and it is rare for it to be seen earlier on the post-mortem table. Yet exceptionally this is the case, and it must be evident that prior to the exudation of any fluid there is a stage of simple engorgement. The air-vesicles would be encroached upon by the distended blood-vessels causing a harsh and deficient natural vesicular murmur. This early condition has been recognised by Stokes and confirmed by Addison, so that it would more justly be styled the first stage of pneumonia, and that to which this name is given would then constitute the second stage. We shall adhere, however, to the usual nomenclature.

*The second stage of pneumonia, that of red hepatization.*—As in the course of inflammatory effusions in other parts of the body the serous exudation at first poured out becomes more solid, so the same occurs in the lungs; the serum makes way for lymph, until the air-cells become completely blocked by it. The lung at this time when cut through no longer exudes serum, but a solid fibrinous matter can be squeezed from the cells; the blood-vessels, however, are still gorged with blood, which is in part stagnant within them; at the same time the secretion which has been poured out contains some blood-globules and hæmatin, and thus the organ still retains its red colour, though not so bright as in the healthy state. This dull-red solid lung resembles, under these circumstances, a piece of liver, and thus the term hepatization has been given to it. On examining the cut surface it is seen to be slightly granular from the projection of the distended air-vesicles; it no longer emits any fluid or air on pressure, but rapidly sinks in water. It is also very lacerable when the finger is thrust into it.

*The third stage, that of grey hepatization.*—This is a further development of the previous stage by a more complete solidification. The air-vesicles are more tightly filled with inflammatory matter, the cut surface is still granular, sinking in water and easily lacerable; but the exudation contains less colouring matter, and the blood has been squeezed out of the capillaries, and thus the whole colour of the lung is paler. The grey colour, indeed, which the lung presents is due mainly to the natural pigment which it contains, and thus it happens that a hepatized lung is much darker in aged persons, where more natural pigment exists, than in children, where pigment is almost deficient; the hepatized lung of children is therefore white or yellowish white. The nature of hepatized lung can be readily understood if we can suppose the minute bronchial twigs and air-cells tightly filled with albuminous material, and, indeed, it very much resembles the preparations in our Museum where the organ is injected with tallow. When a lung is solidified or hepatized in the manner spoken of, it is seen filling the chest when the body is opened, as, instead of collapsing as a healthy lung should do, it forms a mould of the thorax, and when removed the form of the ribs may be seen impressed upon it. Not uncommonly a slight pleurisy has accompanied the pneumonia, and then a delicate layer of lymph may be found on the surface of the lung and easily separable from it. A lesser amount of pleurisy is very commonly present when the membrane is seen to be opaque, whitish, or to have lost its lustre. This is so common that it is only when a considerable amount of lymph has exuded from the surface that the name pleuro-pneumonia is usually given. In comparing the solidified with the healthy lung it will be found that in some cases as much as two or three pounds of solid matter must have been rapidly exuded into the tissue. During life in this second and third stage of pneumonia there is an absence of all natural breath-sound, but in its place there is bronchial breathing and bronchophony with dulness on percussion.

We will now say a few words on the *histology* of the process which takes place in inflammation of the lungs. This, as might be surmised, has varied with the prevailing views regarding the exact changes which take place in inflammation wherever occurring. Before the microscope was employed pneumonia was regarded as an exudation of lymph into a supposed parenchyma of the lung, but when the organic nature of an inflammatory product was discovered by Schwann his theory was applied, and the ordinary explanation of a primary hyperæmia, followed by an exudation of serum called "cytoblastema," in which cells sprung up, was adopted. This theory of a spontaneous generation of cell-life continued until Virchow's dogma of "omnis cellula e cellulâ" was enunciated, and the idea of proliferation of the endothelium of the alveoli of the lung was substituted for the older one; on this theory the hyperæmia must be regarded as secondary and necessary to the cell-growth. More recently the views of Cohnheim have been enforced by many pathologists, that inflammatory cells are none other than the white cells of the blood which have migrated through the capillaries, and these views may be made applicable to the case of pneumonia. It does, however, appear to be a fact beyond contradiction that in pneumonia the endothelial cells grow and propagate, but this by no means is opposed to the later theory. If the air-vesicle of a case of so called croupous pneumonia be examined, it will be found to be filled with a firm and fibrillated substance, in which are embedded cells resembling the white cells of the blood, and others larger and of less regular shape, which may well have been formed by the proliferation of endothelium; at the same time, owing to the engorgement of the vessels, some red corpuscles have escaped into the tissue, and some of them breaking up have stained the secretion red; this is observed in the rusty viscid sputum. At the later stages of pneumonia we may also observe the fibrous walls of the alveoli containing an exudation of cells, and these may in all likelihood have had their origin in the fibres themselves. It has long been a subject of controversy as to whether the bronchial or pulmonary arteries mainly share in the inflammatory process, and opinions are still divided on the point. It cannot but be observed, although the term inflammation is used, how an extreme blood-pressure might sufficiently account for an exudation of fibrin, with red and white corpuscles and desquamating epithelium.

*The stage of purulent infiltration.*—According to the view which may be taken of this condition it may be regarded either as a necessary sequel to the forms of inflammation just described or as a true fourth stage of pneumonia; indeed, if the hyperæmic state be regarded as a recognisable condition, the purulent infiltration would constitute a fifth stage. It is better, however, to be content with the three which are usually adopted. With the third stage, or that of grey hepatization, the inflammation has reached its height, and the attendant fever is departing; resolution takes place, the exuded lymph undergoes disintegration, the cells become granular and fatty, and thus the inflammatory products are removed by absorption or expectoration. The alveoli are surrounded by lymphatics which, no doubt, carry oft this liquefying material. They may be seen of large size on the surface of the lung and associated with the swollen glands at the root. If then a simple absorption takes place, the exuded matter may be taken up as rapidly as it was thrown out; this is certain from the frequent absence of expectoration and the return of the diseased lung to its healthy state. More frequently the secretion is thrown up, and the subsidence of the inflammation is shown by the change of the rusty viscid mucous to a thin and yellowish one. It is during this period, when the exudation is softening and passing into the tubes, that pneumonia is usually fatal, and it is frequently observed that the sputum instead of becoming lighter in colour has become purple or black. When a post-mortem examination is made and the lung said to be in a state of purulent infiltration, it is found not to have lost its solidity, for it will still sink in water, but when cut through, instead of being dry, it exudes a dirty-coloured fluid of a greenish-brown colour mixed with air. It is the hepatized lung softening and saturated with the fluid products; the exudation-cells are granular and broken up, and the alveolar walls full of the same material. Considering that in fatal cases of pneumonia this is the condition found, the stage of purulent infiltration has been regarded by some as a necessarily fatal one,—that instead of a simple disintegration of the cellular exudation taking place as in the progress towards recovery, a further infiltration occurs in the alveolar walls, with a loss of elasticity and actual softening, rendering recovery an impossibility. Yet there are those who with Hughes Bennett regard a change resembling this as a necessary termination of an inflammatory process, and therefore a purulent stage of pneumonia as an all but necessary one. Of course he would not imply, nor is it implied, that in the fatal form the term purulent is equivalent to the word suppurating; it signifies merely that the inflammatory cell or leucocyte becomes granular and fatty, and as some would further say, a pus-cell, but it does not mean that anything like purulent matter is seen in bulk or that abscesses are ever formed. Suppuration, in the ordinary sense of the word, does not take place as one of the stages of inflammation of the lungs. If an abscess is found in the lung the inflammation has not been of the ordinary or croupous variety, but it has been lobular and pyæmic. If not it must be most exceptional.

It is a point of great interest to ascertain when the recoverable stage of pneumonia has passed and the fatal one begun. It may be true that the disintegration of the material is necessary, and thus theoretically a purulent stage must exist; but, as a matter of fact, mucous râles throughout the chest are regarded as unfavorable during the recovery of pneumonia, whilst the most favorable cases are those where absorption quietly takes place without any moist sounds whatever. These are the reasons against thinking that the purulent infiltration usually found in fatal pneumonia is a stage which is reached in cases which recover; the probabilities are in favour of the hepatization in most cases not even progressing much beyond the red stage.

The micrococcus found in the lungs in pneumonia is peculiar in being enclosed in a capsule, which is rare in other forms. It is called the *pneumococcus.*

*Lobular pneumonia.*—This may occur in connection with obstruction of the pulmonary arterioles, as in pyæmia, and therefore is not a primary affection of the lung; it may also occur in connection with bronchitis, a form of malady mostly met with in children; and thirdly, it may be seen as a chronic affection laying the foundation for pneumonic phthisis.

*Phlebitic and pyæmic pneumonia.*—This is limited to individual lobules, or a group of them, their number being determined by the size of the pulmonary artery which is primarily involved, the peculiarity of this form of inflammation being its rapid progress, and its termination in abscess, or sloughing. Its most frequent seat is the lower edge of the lobes, especially the inferior ones, where firm masses of consolidated lung may be seen, each mass averaging in size that of a walnut, although it may be greater or less. On cutting through these masses the various stages of inflammation may be seen, one perhaps being in a state of red and another of grey hepatization, and another of abscess; thus these stages occur in rapid succession, and in the more favorable cases in the order already mentioned; and where recovery takes place, which is probably very rare, merely the hepatized state has been reached; but as a rule the disposition is to rapid disintegration, and thus within two or three days after the first onset of inflammation symptoms of sloughing or suppuration occur, the tendency being always to form an abscess. The disintegrating process probably comes on very soon after the first inflammatory stage; and no sooner is the exudation poured out than a rapid breaking down of the tissue ensues, and thus the lung may be found softened or sloughing with the surrounding parts only in the first stage of inflammation. It is worthy of remark how this form of disease occurs near the surface of the organ, as it does also in the liver, &c.; and the same is true also, though to a less extent, of adventitious products in general, the cause of this distribution not being positively made out. If the first process be infarction it may be due to the edge of the lung having less egress for escape of blood than a portion of its middle. Since this is the case, the pleura becomes involved in the inflammation, and thus every diseased portion of tissue is covered with a layer of lymph; and in some cases the whole lung is thus affected, or a general acute pleurisy has taken place. In this instance the abscess has generally burst into the pleura and so caused the serous inflammation. This form of disease, or lobular pneumonia, is generally connected with purulent absorption or infection, and is thus most frequently the immediate cause of death in pyæmia arising from injuries, operations, &c.; it was once thought that the pus was actually carried from the distant part to the central organ, and if no pus were present there, it was formed in the vein by phlebitis; but the disproof of this is, that all the stages of inflammation preceding the production of pus are present; there can be no doubt that something is carried to the lung from the infected part, and although this is not actual pus, it is probably some of the elements of pus or other infecting matter, for experiments have shown that the disease is produced after injecting such like material into the blood, and is probably due to a specific micrococcus. When this has been done, the immediate effect produced is a coagulation of the blood or infarction; or, if smaller quantities of infective matter be used, it permeates into the smaller vessels, and there produces its effects, the first indication being a spot of congestion from coagulation of blood in the minute pulmonary arteries; thus, on making a section through a lung the subject of lobular pneumonia, we find, as we have already mentioned, the various results of inflammation; and preceding these, parts of the lung of a red colour; these patches of congestion being the first step in the process, and since in each mass the more rapid changes are towards the centre, an abscess may be found in the midst, the surrounding part hepatized, and the circumference of a red colour from this congestion of the tissue. It is highly important to notice these congestive patches, for we believe they always show blood disease, as already mentioned under fever and purpura; and in cases of purulent infection they precede the further inflammatory changes.

On opening the pulmonary arteries the blood is found coagulated and the vessels plugged. In some cases it would seem that the fibrin had formed primarily in the larger trunks, and then being disintegrated had been distributed through the smaller branches, thus blocking them or setting up several centres of infection. In other cases it would seem that some deleterious matter circulating in the blood promotes its coagulation in the extreme vessels themselves. In whatever way the infarction occurs the fibrinous plug acts as an irritant, and sets up an inflammatory action in the lung tissue; at the same time, cutting off the blood supply, it favours a rapid sloughing, or even necrosis of that portion of the lung to which the blocked arteriole proceeds.

*Catarrhal or broncho-pneumonia.*—This is a very important affection, and constitutes for the most part the inflammation of the lungs of children. Children are not exempt from lobar pneumonia, but are more commonly affected by a bronchitis, which is apt to pass into the lobular and vesicular form. The inflammatory process extends down the tubes into the lungs, and at the termination of a certain number of bronchial twigs one or more lobules become affected, an exudation is thrown out into the cells, and the tissue becomes consolidated. If death has occurred at an early stage, and individual scattered lobules are alone invaded, the affection may be overlooked. If the lung, however, be carefully examined, numerous small portions of tissue may be found airless and of a redder colour than the surrounding parts; some of them may be merely collapsed lobules, but others will be found to be soft and granular, and exuding some lymph when squeezed, which again shows abundance of inflammatory corpuscles when examined by the microscope. At a later stage the inflamed portions become lighter in colour, and then may be seen on a section of the lung a number of small white bodies like millet-seeds; these are the lobules filled with exudation, and are likely to be called tubercles by the inexperienced eye; indeed, such vesicular pneumonia, when occurring with disorganisation of the lung in phthisis, is, frequently, called tubercle. When larger portions of the lung are affected a lobular pneumonia is sufficiently evident. The diffused character of the disease does not strike the eye so much as a uniformly consolidated lung, and thus its severity may be overlooked; but all the bronchial tubes should be opened out so as to examine the membrane or the secretion upon it; afterwards the tissue should be looked at, which by squeezing and close inspection may be found in parts airless, and in parts containing an inflammatory exudation. Pleurisy does not accompany this form as often as the pyæmic.

Of late years microscopic examination is thought to have shown a different histological process between the two forms of pneumonia; in the one case corresponding to the croupous exudation in the air-passages, and in the other to the ordinary corpuscular secretion. So that in this lobular or broncho-pneumonia, of which we have been speaking, the air-cells, instead of being occupied by a fibrillated firm mass, are filled with corpuscles or leucocytes, which have had their origin in part from a proliferation of endothelium, in part from the migration of cells from the blood-vessels, and in part from secretion which has been sucked in from the bronchioles and plugged the alveoli of the lung tissue.

It is not absolutely correct to say that there is no fibrillation in lobular pneumonia as it is sometimes seen. Charcot and his followers believe that the walls of the bronchioles are the seat of much of the cell-formation in this and in the more chronic lobular inflammation, and they use the terms peribronchial nodule and peribronchial abscess to designate these formations.

A good example of the affection is met with in measles; its occurrence does not seem to depend upon the fact of the greater frequency of the exanthem in children, for it is met with in a most perfect form in adults. Under exceptional circumstances there may also be met with in adults forms of bronchitis which, spreading downwards, culminate as broncho-pneumonia; thus in tracheotomy and injuries to the air-passages this form of inflammation may be occasionally met with.

*Chronic catarrhal pneumonia.*—In connection with bronchitis in adults we occasionally find that after the disease has existed for some time a certain number of lobules may become affected, more especially at the base of the lung; the consolidated lobules form foci, from which the same process proceeds, until a large portion of lung has become hepatized. These portions being completely blocked up by secretion become hardened, and then undergo further changes. A section of the lung would be found to have scattered through it a certain number of yellowish-white masses of this hepatized tissue. In course of time, if resolution does not take place, these portions become still more dry, the blood supply is cut off, and they are converted into cheesy masses; these subsequently disintegrate or soften, and the lung tissues decaying with them disorganisation of the lung ensues. This epithelial proliferation, caseation, and destruction constitutes *one form of phthisis*, to which we shall presently refer. It is a question whether these caseous masses are the result of a chronic or slow albuminisation of the tissues, or whether they result from a lobular pneumonia which was in the first place acute.

In all probability the *peripneumonia notha*, or bastard pneumonia, a disease about whose nature as described by the older writers there has been much controversy, was, like this one of children, a catarrhal or broncho-pneumonia, and thus we may notice in morbid anatomy, as well as in other relations, how senility is second childhood. The cases so called were those where a sudden and difficult breathing came on, which was speedily fatal, and evidently denoted an obstruction in the pulmonary tissues; and where the post-mortem inspection revealed only a bronchitis, or no evident consolidation of the lung. A careful examination of the tissue in such cases, however, shows that the inflammation had crept down to the vesicular structure which terminates some of the bronchial twigs, and that these had been rapidly filled with exudation; at the same time other portions had collapsed from the bronchial obstruction, and then had ensued the fatal symptoms and rapid death.

Probably even less than this might be apparent where the case was that of a very old and feeble person occurring in the season of winter. For it may be observed, both from the depressing effects of cold on the nervous system as well as from its directly injurious effects on the lung, that the lung will collapse in large portions without any great evidence of bronchitis, and then assume the appearance already mentioned under the apneumatosis of childhood.

*Typhoid pneumonia.*—It has been thought by some pathologists and writers on fever, especially the German, that in typhoid fever the whole body is peculiarly affected, and that various organs especially suffer. The ileum is the part where the diseased condition more particularly exhibits itself, but that all other organs participate in a morbid state, and especially the lungs, is recognised by the dyspnœa, cough, sanguineous expectoration during life, and the congested or inflamed condition found after death. The question is whether this affection of the lung is peculiar—that is, is it due immediately to the typhoid process which shows itself in so characteristic a manner in the intestine—or is it merely due to the state of fever, and the consequent alteration of blood and tissues of the body? It is said by those who believe the pneumonia to be peculiar that the microscope shows characteristic cells of the same form as those in the ileum and mesenteric glands; but then, in objection to this, it may be said that, admitting the specific character of the intestinal affection, it is not satisfactorily shown that the deposit affords any peculiarities when viewed by the microscope. If, then, there be doubt about the microscopic characters of the elements of the typhoid deposit in the intestine, and we trust rather to the naked-eye appearance for its recognition, how much more difficult is it to speak of the peculiarity of a similar affection of the lung when the outward appearances are not so distinctive? It is thus thought by some, and with good reason, that the form of pneumonia found in typhoid fever is due merely and necessarily to the state of blood, tissues, and position of the patient. It is an appearance important to recognise, for though not, we believe, characteristic, yet, when associated with other conditions, assists in marking the disease.

Of late a true typhoid germ has been found in the intestine, but we are not aware of its recognition in the lung, or even in the blood. In all fatal cases of typhoid fever the lungs are found gorged with blood of a dark colour, and present a spotted appearance, both externally and internally, as we have just mentioned is generally the case in blood diseases; it is a stage beyond this, however, which constitutes inflammation, but so intimately are the two stages connected that the one gives the other its peculiar characters, and, indeed, the general appearance of the organ and the part affected show how much the morbid state is due to a mere blood disease. Thus, as the congestion and ecchymosed appearance occupy the back part of the lung, so is the pneumonia found in the same place, and it is therefore at the posterior part that the consolidation, if any, is seated; it is not hepatization of one lobe or another, as in ordinary pneumonia, but the posterior edge, and especially the part near the root of the lung, that is affected, and it seldom passes beyond the stage of red hepatization; this is not uniform, but occurs in patches, and thus, if a section be made, it appears as if a number of lobules had been separately inflamed, leaving some healthy portions between them, or a certain number of lobules in a complete state of hepatization, while the tissue between is recently and acutely inflamed. If the inflammation extend beyond this, and affect a larger part of the organ, it may be looked upon as a complication, in the same way as an ulceration of the intestine following the typhoid process in the ileum. The reason, as we have said, why we are not sure that this condition is peculiar to typhoid, is that it may be sometimes met with in typhus, and other specific diseases as anthrax. Also, as regards the enlargement and softening of the bronchial glands before noticed: this has been considered by some as a peculiarity of the affection; but certainly it is not so, for they are generally found affected in all cases of pneumonia, and more especially where a large part of both organs is involved.

We have here been speaking of the pneumonia which accompanies typhoid fever, but under the name "typhoid pneumonia," a form of complaint has been spoken of suggestive of a pneumonia being the principal feature of the fever during its whole course. That is, that the causes which usually induce a typhoid or enteric fever may, in some cases, alight on the lung, and thus a pulmonary complaint rather than an enteric be the main feature of the disease. The theory is made to account for outbreaks of endemic pneumonia in places of bad sanitary repute.

*Hypostatic pneumonia.*—This is a term used to express that condition of the lung which is so frequently seen in the post-mortem room, affecting their posterior parts. We see, almost every day, lungs removed from the body which at their front parts are dry, spongy, and natural, but having the back parts doughy, heavy, and when cut exuding serum; this condition, conjoined with increased softness of texture, is that which characterises inflammation, and therefore the term pneumonia is adopted; as, however, it does not come on until the patient is dying of some other disorder, it is called *pneumonie des agonisants*, or *pneumonia morientium*, and must be looked upon rather as a result of the act of dying than a cause; and this is not difficult to understand when we consider the prone position of the patient, without any movements for days, and a disordered state of blood, as well as a commencing decay of the tissue, for we have already seen how, in fever, the back part of the lung, from a mere mechanical reason, is the part most liable to the morbid change; and then, if to these be added the decaying powers of the brain and nervous system whereby the influence of the pneumogastric nerve is weakened, we gain some idea as to the cause of this process. In some cases we find the pneumonia has reached the stage of hepatization; and then a question arises whether this be the same form of disease further developed, owing to some accidental prolongation of the patient's life, or whether there be any especial exciting cause for the inflammation: if the pleura be affected, we should have no doubt in saying that such is the case. Thus, in Bright's disease, where an early condition of pleuro-pneumonia is so often found unexpectedly, there is a special cause for its production, and more than can be found in the mere act of dissolution.

*Gangrene of the lung.*—This may occur under various circumstances and to a variable extent. It is usually divided into *general* and *local*, or *circumscribed*, although these do not actually correspond to many cases frequently met with. The former term is intended to apply to a general gangrene following inflammation, whereas the local arises from an accidental death of a particular part. In the majority of cases, however, which we see in the post-mortem room, it arises from the lung being involved in the disease of a neighbouring organ; thus, in cases of cancer of the œsophagus, we may find the adjacent parts of lungs, those near the root, in a state of hepatization or purulent infiltration, and, at the same time, sloughing or gangrenous; we see, on section, a quantity of green, dirty fluid exude, the tissue broken up in shreds, and the odour indescribably fetid. This we know by handling such an organ, and one finds the odour attaching for many hours to the hands, and very different from the ordinary smell of decomposition. In cases of aneurysm of the descending aorta the same may occur from pressure; and we have seen it, two or three times, result from caries of the spine. We must not necessarily expect gangrene of the lung because there has been fetid expectoration during life, for this may occur in connection with bronchitis, the odour being due (so Dr. Laycock states) to a compound of butyric acid which is formed in the lungs. In the *general form* of gangrene an ordinary idiopathic inflammation has preceded,—a pneumonia, in fact, has run on to gangrene. In other parts of the body, this is one of the ordinary consequences of inflammation, but it is so rare in pneumonia that we did not mention it among the usual terminations;

it does, however, sometimes occur, and generally arises from some constitutional cause or epidemic influence; thus, we do not think we have met with a case for a considerable period, although a few years ago we had several such cases, but could not account for the occurrence except on the supposition of some epidemic as influenza; in these the pneumonia rapidly passed into gangrene. In such cases the dead part passes by insensible stages into the healthy, there being no distinct boundaries between them; the most affected parts being soft, shreddy, and having a horrible odour, while the tissue around is soft and exudes a dirty-coloured fluid. Where from aneurysm or pressure of a tumour on the lung, a pneumonia has been set up, a question has arisen as to whether it is due to pressure on the pulmonary plexus of nerves affecting the nutrition of the lung, or to the increased and retained secretion in the bronchial tubes, which at last infects the pulmonary tissue. In the *circumscribed form* of gangrene a portion of lung is found isolated from the surrounding parts, and is dead; this may result from a simple inflammatory process, and is due, no doubt, to the stoppage of the blood-vessel proceeding to it. This may arise under various circumstances. The thrombosis may be infective and connected with ulcerative endocarditis or other sources of infection. Sometimes it may occur in old age from senile change in the blood-vessels; at other times it may be associated with dilated bronchi; the sacculi being full of fetid contents with ulcerated walls and gangrenous condition of neighbouring pulmonary tissue, or an apoplectic portion of lung due to infarction may be cut off and die. No cause, however, is more likely to produce gangrene of the lungs than irritation of the smaller bronchi set up by the introduction of foreign bodies into them. We have seen, in a case of disease of the larynx, imperfectly chewed food continually passing into the air-passages; the consequence was the production of gangrene scattered through both lungs.

*Chronic lobular pneumonia.*—This is the disease already alluded to where, in connection with bronchitis, certain groups of lobules become consolidated and converted into yellowish-white masses. If they soften and cause disintegration of the lung they constitute one form of phthisis—the acute pneumonic phthisis of some authors.

*Local indurations of the lungs.*—The nature of these will be further discussed in the next paragraph, but it may be here stated that in various forms of chronic disease of the lungs these indurations are met with; and in phthisical lungs the cavities are surrounded by dense tissue. Those who are in the habit of making post-mortem examinations know how common it is to find consolidation of the apices which are at the same time adherent to the chest wall by thickened pleura. It would seem as if the connective tissue of the lung, together with the walls of the air-cells, had undergone an hypertrophy whereby the alveoli had become obliterated, and the true pulmonary tissue converted into a tough fibrous substance. On cutting through these indurated portions of lung they are dense and quite airless, and so exceedingly hard that it is impossible to thrust the finger into them. It may also be observed that wherever these indurations occur the tissue is darker than that of the surrounding healthy parts; this is due to hæmatin from the changes which take place in the blood during the inflammatory process. The microscope shows how the whole of the lung tissue is united together in one mass. In many cases, judging from the history, the indurations and cicatrices had had a pyæmic origin.

*Chronic interstitial pneumonia and cirrhosis of the lung.*—There is no piece of pathological anatomy about whose true nature so much controversy has been aroused as that of chronic affections of the lungs, one main question being whether the different appearances met with, often in the same lung, are essentially of one kind, or whether they are due to two or more causes, since different observers may see in the same lung tubercle, scrofulous deposits, caseous changes of inflammatory products, true interstitial pneumonia, and cirrhotic processes. It is therefore necessary to speak separately of the conditions known under these names.

As distinct from the more ordinary pneumonic changes seen by a cell-production in the alveoli, and even from the more chronic forms of a similar character, there is a condition tending to induration of the tissue where a production of fibrin or fibro-plastic material swallows up and envelops the whole respiratory portions of lung. The term interstitial is scarcely correct, as it might imply a process limited to changes in the interlobular connective tissue, whereas the process of which we speak commences in the alveoli and their walls. A portion of lung is seen to have entirely lost its spongy character, and becomes hard, dense, and fibrous; also, owing to the presence of pigment, it was styled by Addison iron-grey induration, and he also likened it to Aberdeen granite. The microscope shows a fibro-nucleated material occupying nearly the whole of the pulmonary structures, although within the alveoli there may also be found cells and nuclei. It is a question whether the new product has its origin in the fibrillation of a cell-formation, or whether it does not spring up entirely in the walls of the alveoli and blood-vessels, and then slowly proceed after the manner of a growth. This indurated fibrous tissue sometimes puts on a granular form, and thus it has often been described as associated with tubercle, and, further, it has been maintained that the grey tubercle of fibro-cellular formation has a pathological and histological origin perfectly similar to that of this new tissue, and may have given rise to it.

It is not uncommon to find lungs indurated in this manner throughout a large part of their volume, and to this the name chronic pneumonia is usually applied. In some cases, as above mentioned, the surface is granular, and to this the name tubercular is given; in other cases distinct cheesy masses are interspersed with the fibroid material, showing that a corpuscular inflammation has once been present in the air-vesicles. Sometimes the tissue ulcerates and cavities are formed, and to this condition the term chronic pneumonic phthisis is by some applied.

Sometimes with this dense nucleated fibrous tissue, closely interwoven with the alveoli, the bronchial glands have been changed into a similar hard and fibrous substance. In some cases the bronchial tubes are much thickened, giving rise to the opinion held by some that the coats of the tubes are the seat of many of these new productions, and they would name the disease a peribronchitis. There does, however, seem to be such a disease, though rare, of a simple acute interstitial pneumonia. In a patient who died of bronchitis and emphysema portions of lung were very hard, and were found to be involved by a tough fibre tissue swallowing up the air-cells. These hard portions did not appear as circumscribed masses, but as streaks and bands invading the pulmonary tissue. To such a condition, when far advanced, the term fibroid disease would be applicable.

An extreme form of the indurated lung which we have been describing is known by the name of *cirrhosis*. Many pathologists have been unwilling to separate it from the former, or chronic pneumonia, but have been generally forced to do so from the fact of the disease being so much more complete, and from often being confined to one lung, and unaccompanied by any changes in other organs. The chronic pneumonic or fibroid disease is never confined to one lung; it is therefore called phthisis, and has a constitutional origin, whereas the cirrhotic disease has often a mere accidental cause for its beginning. In a well-marked cirrhotic lung the organ is shrunk into a small compass, with all or nearly all the respiratory spongy tissue destroyed, all being swallowed up in a dense fibrous tissue. This is so dense that it cuts like fibro-cartilage, showing a surface smooth and grey, which is generally intimately blended with an enormously thickened pleura, which also cuts like cartilage. In the majority of cases the section shows the lung permeated with thickened bronchial tubes, many of which are also immensely dilated so as to form large cavities. There may be distinct excavations in the tissue also. The main peculiarity of the disease is the fact of the conversion of the spongy lung into a dense mass of fibrous structure with enormously dilated bronchial tubes running through it. There has been much controversy as to the relation between the dilatation of the tubes and the induration of the tissue. Laennec believed, and he has still many followers, as Wilson Fox and Fagge, that in cases of enlarged tubes with a dense intervening tissue the cause was primarily in the bronchi, which being inflamed, a softening of their walls occurred, and a giving way of their boundaries, with a necessary compression of the intervening tissue. Corrigan subsequently gave his opinion that an inflammation and contraction of the pulmonary tissue constituted the primary change; that in consequence of the contraction of the tissue the tubes became expanded as a compensatory result. In favour of the view that the cirrhotic process is one of the tissue and altogether local is shown by the fact, which in some cases seems undoubted, that it has begun as a pleurisy; the pleura has slowly become thickened, and at the same time the fibrous thickening has continued inwards through the whole lung until it has become universally indurated. Sections of lung may show the fibrous element surrounding the bronchial tubes and the blood-vessels, and the walls of the air-cells thickened until the whole structure of the lung is involved in the new fibro-nucleated tissue. It seems to have been a true interstitial pneumonia without the occurrence of any more active form of inflammation. Those who argue for the unity of phthisis, and that all disorganising diseases of the lung have their origin in tubercle, would include cirrhosis in the category, and maintain that a part of the process was tubercular even though a part were inflammatory.

*Other forms of chronic pneumonia.*—It will be presently seen under the head of phthisis how opinions vary as to the nature of many of the chronic formations found in that disease. There are those who would regard them as varieties of chronic pneumonia, and would therefore rightly describe them in this place. It will be observed that under the heading acute pneumonia a lobar and lobular pneumonia have already been mentioned, and that the latter may be of the chronic form; and also that there is a chronic pneumonia whose nature is believed to be of a totally different kind, being, indeed, the result of a fibroid and interstitial change; but it remains for further discussion to ascertain whether there is not a chronic pneumonia whose nature partakes more of the ordinary lobar, or so-called croupous kind and developed from it.

We refer now to cases where, after an illness of a few weeks, during which time all the evidences of consolidation are present, the patient dies, and the lung is found (to use Addison's expression) uniformly albuminised. The section is not soft, lacerable and granular, as in the acute grey hepatization, but it is smooth, solid, and tough. The air-cells are full of fibrillated lymph, and the exudation-cells may have commenced to be granular and fatty. It is a condition of lung very liable to break down, and thus constitutes a case of rapid phthisis, and on the other hand it is a condition from which recovery may take place, and the lung be completely restored. That there is a chronic pneumonia of such a kind can scarcely be denied when it is remembered for how long a time all the signs of consolidation may endure, and then a complete restoration take place. We must therefore believe that there is a true chronic pneumonia, as Charcot describes, whose origin is an ordinary inflammation and exudation into the alveoli, and whose appearance is best denoted by the term *uniform albuminisation*, remembering that, although occupying a large part of the lung, it may be essentially of the chronic lobular kind, in which the various foci of inflammation have run together. We believe, however, the lobar form is rare, for as a matter of fact an acute pneumonia either resolves itself or is fatal in a few days. The continuance of physical signs for a longer time, which was formerly regarded as evidence of the perpetuation of the consolidation, is due rather to the pleurisy which accompanies it. It is right, however, to state, as before said, that there are good observers who maintain that acute pneumonia may become chronic, though they admit the rarity of the occurrence.

*Phthisis.*—The pathology of phthisis has been undergoing a complete subversion since the discovery of the tubercle bacillus by Koch. As this, however, does not affect the question of the anatomical changes which take place in the lung, we shall discuss these as heretofore, although the presence of these microbes must necessarily have a direct bearing upon the mode in which we regard these morbid products in the lung. The term phthisis is generally used to include all chronic destructive diseases of the lung, and is equivalent to the popular expression "Consumption." If the limitation of the term tubercle be made to depend upon the discovery of bacilli, then there are forms of phthisis, according to some, which are not tubercular, as for example, the most acute form of an inflammatory character, as well as the most chronic kind known as fibroid phthisis. All forms, however, according to some, are tubercular and bacillary. In a large number of cases it is true that the destructive changes in the lung appear to be due, in the first place, to the formation and subsequent disintegration of tuberculous matters which have been formed in the air-cells. This has been associated with some ordinary inflammatory products which have taken part in the destructive process; although they have also been instrumental in the process of repair, as seen in the large amount of fibrous tissue hardening the lung and circumscribing the cavities and binding them to the chest.

Tubercle may be found as a firm translucent body, scattered through the lung and called miliary tubercle, as well as a softer yellow material called by some crude tubercle, and the relation between these two has long been a subject of controversy; some indeed denying that the latter deserves the name of tubercle, being nothing more than a degenerating inflammatory product. Laennec, who was the first systematic writer on the subject, described the miliary tubercle as the primary formation, and that this afterwards changed to the yellow, softer form; he also spoke of tubercular granulations. Taking the word tubercle in a not too restricted sense, it is found when appearing in the lung as a nodule composed of several alveoli filled with cell-elements conglomerated into a roundish mass. It is composed of cells, nuclei, and polynucleated cells or giant-cells. The cells are of irregular shape, and raise a doubt whether they should be called cells or nuclei; the giant-cells when present contain several nuclei. These single and multiple cells are so little characteristic that some pathologists maintain that tubercle cells are no more than altered leucocytes, and they also maintain that there is no essential difference between the two forms, although they have not decided whether they take their origin in the epithelial lining of the alveoli or the connective-tissue corpuscles. Those who make varieties of the two forms of tubercle describe the miliary as having its origin in the alveolar wall, and composed of cells contained in a fibrous matrix or reticulum, whilst the yellow arises in the epithelium of the alveoli and termination of the bronchi, and is composed of a mass of irregular-shaped cells consisting of epithelium and lymph-corpuscles. These become opaque and fatty and block up the air-vesicles.

Tubercle has been thought by some to have a resemblance to a lymph-gland structure, and has been called therefore adenoid or lymphoid; indeed, it has been thought to have had its origin in the lymphatics. In some parts of the body it is clear that tubercles have their origin in the coats of the blood-vessels, and in phthisis this has been shown to occur by Dr. C. Turner, who had a specimen where the pulmonary artery and its branches were covered with minute tubercular granulations. Some few years ago, the non-specific character of tubercle was supposed to be proved by the capability of inoculating rabbits from the inflammatory products of a sore; if these were caseating and introduced into the skin, a number of like products were found scattered through the body and called tubercles. A caseating gland therefore was regarded as a possible source of infection, and equal to the production of a general tuberculosis. The discovery of the specific bacillus naturally threw a suspicion upon the accuracy of these experiments, and subsequent researches have shown that sufficient care was not taken to separate true tubercle from the simple products of inflammation. The latter are now said not to be infective. The modern prevailing view is that true tubercle must have a specific bacillus for its source. It is surmised that the bacillus is taken into the lungs either through the inspired air or through the blood-vessels,—that, penetrating into the alveoli, it excites the multiplication and growth of cells in their walls or within their interior. Some have hazarded an opinion that the form is miliary when the passage of the microbe is by the vessels, and that it is yellow when the microbe is inspired through the air. Other products of an inflammatory kind may be found mixed with them, so that on examination of a phthisical lung there may be found a certain amount of solidification due to hepatization; also firmer masses of albuminous material due to a slower inflammation, as well as a dense fibrous tissue which conglomerates all the structures of the lung together. There are some pathologists who do not see the necessity of introducing the question of ordinary inflammation, believing that the tubercle itself may undergo further development, and grow into large masses of granular fibrous structure.

Addison taught that the only true tubercle was the miliary, which never altered, and that the destructive process in the lung was due to the breaking down of the yellow material, which although usually called tubercular Addison believed to be degenerating inflammatory matter. Niemeyer followed in the same road, maintaining that the destructive processes in the lung were due to inflammation, and, moreover, that acute phthisis was purely inflammatory, and resulted from the broncho-pneumonia already spoken of, that a proliferation of endothelium took place at the end of the smaller bronchi as well as in the alveoli, and that the cell-mass formed there underwent caseation and subsequent disintegration. Whatever view be taken, this morbid material softens and destroys the lung tissue until a small cavity is formed. Many of these run together producing a large vomica.*

If we take a large number of phthisical lungs and regard them as a whole, we shall perceive how all these products of which we have been speaking are intimately associated, and how it has been easily suggested to the observer to look upon them as but different forms and stages of one simple pathological process either tubercular or inflammatory. We may see, for example, a lung considerably disorganised the subject of acute inflammation; another in which there are albuminous deposits not distinguishable from what are seen in the different stages of hepatization; and another in which there are undoubted inflammatory products associated with the before-named yellowish deposit whose origin is disputable, and with these perhaps also miliary tubercle. In all these varieties we may see also fibrous thickening, and indurations of a chronic kind.

*General appearances.*—In a typical case of phthisis in a young person, which has existed from one to two years, the lung as a rule has been first affected at the apex, and the disease has gradually progressed downwards, usually in one lung in advance of the other. Consequently the greatest disorganisation is found at the upper part whilst the deposit which has constituted the origin of the disease is seen as a recent formation in the lower lobe. Whilst destruction has been going on, reparative changes have also been in progress, and, consequently, these are best seen where the disorganisation is greatest, and thus considerable induration of the tissue may be seen at the upper part of the lung, where also the pleura, covering the apex, may be extremely dense while the lower lobe is adherent by soft adhesions. In the upper lobe there may be a large cavity, the extreme apex escaping, its walls dense and adherent to the chest, these walls being composed of thickened pleura and the remains of indurated pulmonary tissue; the lower part also of the cavity next to the lung may be surrounded by a similar hard material. Below this there may be other and smaller cavities of more recent formation, and in consequence the tissue which circumscribes them is not so dense. The cause of the cavities generally involving the upper lobe has not been agreed upon.

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* In the controversies which have taken place as to the tubercular or inflammatory nature of phthisis there can be no doubt that with many the former view supposes a constitutional disease, and the latter a purely local or accidental one. Addison insisted as much upon this great clinical distinction as he did upon the purely anatomical difference of what he considered to be the two forms of the disease. He saw as one form the case of a young person who, in a predisposed constitution, fell an early victim to consumption of the kind where the disease comes on insidiously and progresses from apex downwards, and he saw as another form of phthisis the case of a middle-aged person who, from causes undermining his constitution, such as diabetes, would be liable to a pneumonia, exhibiting itself as a consolidation of a lobe of the lung, and afterwards disintegrating. He might also see the second or inflammatory form not necessarily end in speedy disintegration, but the product, instead of undergoing caseation, fibrillate, producing an induration of the lung. It can scarcely be denied that, clinically speaking, two such classes of cases exist, and therefore those who see in every disorganising lung a material which they distinguish from inflammatory, and call tuberculous on account of its peculiar growth and destructive tendencies, would deprive the word tubercle of much of its original meaning, and make these two forms of disease essentially alike. The controversy would strengthen the opinion that all nomenclature of disease should be clinical rather than pathological. Yet it must be remembered that some of our best pathologists maintain that tubercle is at the origin of every form of phthisis—that rapid consumption means nothing more than tubercle rapidly caseating and disintegrating as the most chronic disease means tubercle which has undergone a fibroid change. Moreover, it is said that in both bacilli are found.

As regards the cavities in the lungs, these vary according to the age of the disease; in the more recent deposit, a mere hollow will be found containing pus and broken-up material; while in the upper lobe, where the disease is older, the cavity may have a hard wall, and have travelled down the organ to some extent. Dr. Kingston Fowler has stated that the primary seat of disease is not the extreme apex but a point about an inch or an inch and a half below this, and nearer the posterior than the anterior surface of the lung. Less frequently it is at the outer part of the upper lobe corresponding to the first and second interspaces below the outer third of the clavicle. He also stated that the apex of the lower lobe is liable to very early infection, as a rule before the opposite lung is invaded, and that from this point there is gradual extension downwards towards the base. Many reasons have been given for the commencement of tubercular broncho-pneumonia in the apex of the lung, among which we may mention the following: the range of respiratory movement is said to be less there than elsewhere in the lung, and the amount of blood in circulation less, and hence deficient nutrition and increased vulnerability. For similar reasons the residues of previous non-infective inflammations linger longest at the apex, and form a suitable nidus for the bacillus, and from sluggishness of the circulation any bacilli that reach the apex are less readily carried off by the lymphatics, and destroyed by the living tissue-cells. During the formation of cavities the bronchi and blood-vessels are differently affected; the latter become closed and sometimes form hard cords which are seen traversing the vomica from side to side, and thus hæmorrhage is precluded. If we cut through one of these, we may sometimes see the obstructed vessel within, thickened externally by indurated lymph, and remains of lung tissue upon it, but often these cords consist of nothing but indurated fibrous tissue. They may, however, be vascular from the formation of new vessels. Louis long ago stated that new vessels were formed in chronic disease of the lungs, and even in the adhesions which bind the lungs to the chest. Whilst the vessels are being obstructed, a different process occurs in the bronchial tubes; these are destroyed, together with the lung tissue, and are gradually worn down even with the walls of the chamber, and so are found opening on its surface, and through these the contained matters are discharged. Sometimes a cavity is round and circumscribed, with hardened walls; at others it proceeds downwards, forming a long straggling space with the obliterated blood-vessels traversing it. As we before said, in the indurated parts of the lungs, the cavities there found communicating with the tubes may have resulted in an expansion of these tubes, although when large it is difficult to prove this; for all trace of the original bronchial mucous membrane is gone, and the cavity could not have been produced without a corresponding wasting of the parenchyma around it, and thus it is often useless to discuss the manner by which such cavities are formed. It is only, however, in indurated parts of the lungs that such can occur, and, therefore, in cases of chronic pneumonic phthisis the cavities found within the lungs are generally expanded bronchial tubes, the indurated tissue being little prone to soften. Old cavities, however, produced by ulceration of the tissue, when lined by smooth membrane and communicating with tubes, may be mistaken for dilated bronchi.

The lower portions of the lung may be occupied by tubercular deposit or so-called cacoplastic lymph, and some of this may be already softening, and small cavities becoming formed. Portions of lung may be occupied also by a morbid product, not in the form of distinct particles, but as a translucent gelatinous material which has been regarded by some as the matrix of true tubercle. In these morbid products the bacilli may be found, and more especially on the surface of the cavities, being more numerous where the disease is actually progressing.

The bronchial glands partake of the nature of the disease found in the lung, and therefore in cases of phthisis tubercle and soft caseating matter may be seen in them. Kidd found bacilli in the bronchial glands in six out of thirteen cases of phthisis.

Having given the above as a typical case, we may mention that a great variety of appearances may be met with, dependent mainly upon the acuteness or chronicity of the disease. Thus sometimes cases of phthisis are met with where the disease has run a very rapid progress; when the destructive process has gone on without any compensating preservative action. There has been no induration of any part by the development of connective tissue, but the whole lung is crowded by soft yellow albuminous or tuberculous matter which has undergone so rapid a softening that the whole lung resembles a sponge soaked in purulent matter. There is no miliary tubercle properly so called, and the only morbid product is the whitish-yellow material which is undergoing caseation. The cavities if formed are ragged and soft, their sides being composed of the disintegrating lung. Towards the bases there may be considerable masses of the morbid material not yet softened, and, according to Addison, not distinguishable from the ordinary grey hepatization of the lung.

We meet also with cases even more acute than this, where the illness has extended over a few weeks only, and has been attended throughout by febrile symptoms. Such cases have been called acute pneumonic phthisis, the only physical signs, in the first place, being those of a pleuro-pneumonia, shown by consolidation of the lung. Subsequently, a breaking down occurs accompanied by the symptoms of phthisis. Addison described several cases in connection with his essays and drawings of phthisis ; for example, that of a young man who had been ill two months from the time of getting cold ; he had high febrile symptoms and all the physical signs of disorganising lungs. The lower lobes of his lungs were found solidified and covered with lymph ; the section showed them occupied by deposits of a low organisable character, pale yellowish, in small circumscribed masses, and breaking down. In two other cases of young girls, with a similar history, the consolidation occupied large portions of the lung tissue, and each albuminous mass was softening in the centre. Such cases Dr. Addison believed to hold an intermediate place between true tubercular phthisis and pneumonia. The more general or lobar the consolidation, the more rapid is the inflammatory process and the nearer is its approach to ordinary simple sthenic pneumonia ; the more it approaches to lobular or vesicular consolidation, the more chronic is the process and the greater resemblance it has to tubercular or scrofulous disease.

In many forms of cachexia and debilitating disease this form of phthisis is met with ; thus Addison's typical case of pneumonic phthisis came from a patient who had suffered from diabetes. The lung had been uniformly albuminised and then broken up into cavities. In these cases of acute pneumonic phthisis, the inflammatory process may still be seen in progress in parts of the lung in the form of small distinct granular masses of grey or red hepatization.

In contradistinction to the last-named form we have chronic pneumonic phthisis, a disease which may have lasted for some years and mostly met with in the later years of life. In these cases the lung may contain cavities and circumscribed yellowish masses of albuminous material scattered here and there, which would be called inflammatory or tuberculous respectively by the advocates of the two theories of phthisis ; but the lung for the most part is indurated, the tissue has become conglomerated into a hard mass by the formation of a connective tissue which binds all the structures together, and which also is of a dark colour from pigmentation. Such a lung is very hard and requires considerable force to be used in order to cut it through. The albuminous circumscribed masses just spoken of may be found also to have undergone a cretaceous change or become calcified.

Where one lung is entirely destroyed in its proper structure by the production of a fibrous tissue, rendering what was once the lung changed and hardened, the name *cirrhosis* has been given, as already said, to this condition. The whole lung has become, in fact, one hard mass of fibrous tissue closely adherent to the chest by thickened pleuræ of the densest structure. In some cases it is evident that the morbid change has commenced in the pleura on the surface of the lung, and the latter has become secondarily invaded. The disease thus appearing to be due in many instances to a local cause has tended to remove it from the class of cases which we have been hitherto considering, which all arise under hereditary or acquired constitutional conditions, and yet in cases where both lungs are involved and no other name than phthisis can be given, the clinical history points to pleurisy as its origin.

We constantly find remnants of disease in the lungs of those we examine, showing that the affection under consideration is curable. What we generally meet with indicating this is the apex of a lung adherent, and its surface puckered, and, on making a section, the tissue is indurated, dark, and only in part permeable to air, and often containing earthy or chalky matter. The probabilities are, that in these cases a deposit of tuberculous matter, with some inflammatory exudation, occurred at the apex, and, instead of softening or increasing, it ceases under some advantageous circumstances, and dries up, but the indurated tissue remains, and with it any earthy constituent of the tuberculous matter. It has been thought by some that this puckered tissue, having often a cicatricial appearance, represents a closed cavity, but there is no proof that this ever occurs. Probably, if a cavity has once formed, it never completely closes. The earthy matter found in the lung is sometimes expectorated during life, as seen in our specimens. Curiously enough, a kind of mould may form in old phthisical cavities, as described by Bennett and Bristowe; this parasitic fungus (but probably there is more than one) is mentioned by Küchenmeister as the *Aspergillus pulmonum hominis.*

In one sense we may find phthisis cured. For example, an upper lobe may be surrounded by dense fibrous structure; itself consisting of a number of cavities separated by similar fibrous tissue. The whole of the morbid material, whether inflammatory or tubercular, has gone, leaving merely holes, and the fibrous tissue encompassing them.

It may be observed that although fatal hæmorrhage is more liable to occur at an advanced stage of phthisis, yet severe hæmoptysis is much more frequent in the early stages of the disease. It then arises from ulceration of some of the smaller pulmonary vessels or from some local congestion of the bronchial arteries. At a later period when cavities are formed the blood-vessels have become sealed, so that a fatal hæmorrhage occurs under other circumstances. This arises generally from the vessels on the walls of a cavity having become aneurysmal. A small sac, the size perhaps of a bean, may have formed on the hardened walls of an old vomica, and then ruptured. The cavity need not be large, but it must be old. Even when no hæmorrhage has occurred, an aneurysmal condition of vessels may be found if looked for. Dr. Fagge had a case where the child was only three years old. Dr. Goodhart once found several aneurysms in the same lung.

**Syphilitic Disease of the Lungs.**—That syphilis will attack the lungs there can be no doubt, but exactly what share it takes in many of the destructive or phthisical diseases is still a question. Distinct syphilitic gummata may sometimes be met with in the substance of the lungs, having the same formation and character as similar deposits in the liver and testes and other favourite seats; the coexistence of these in different organs can leave no doubt as to their nature. The first well-marked case of syphilitic gumma was described by us in the 'Path. Trans.' for 1859. In association with syphilitic deposits elsewhere there were found two round masses in the upper lobe of each lung. One was softening, and appeared to have been deposited in layers, whilst the other was firm and dry. Since this time we have met with cases where there were several defined masses in the lung. The most usual results of syphilis, however, are fibroid thickenings and indurations; these are less characteristic, and hitherto have been included under the general name phthisis. A very remarkable case, in proof of the nature of the disease, occurred under Mr. Birkett, of a woman who had a large syphilitic sore on the thorax, which involved the ribs and penetrated the chest. It was found that the same fibrous substance which formed the floor of the ulcer involved the pleura beneath and the lung itself, so that the latter contained the same fibre tissue as the syphilitic sore. Such a case proved that an interstitial or chronic pneumonia may have a syphilitic origin. In any given case of phthisis occurring in a syphilitic person a question may arise as to the true character of the morbid change in the lung which has led to its destruction, and at the present time tests are wanting to enable us to assert that products, usually called fibroid or tuberculous, have been produced under the influence of syphilis. That such an influence has been exerted is proved by clinical rather than pathological facts, where patients come before us presenting all the usual characters of incipient phthisis or rather pneumonic consolidations, which are speedily and remarkably cured by specific remedies, as iodide of potassium. Under these circumstances there can be no doubt that exudations occur peculiar in their nature and formed under a syphilitic influence, for on no other conjecture can it be explained that they should be so readily removed under special treatment. Whether the morbid products, if they could be then seen, would present any special appearances capable of recognition is a matter still open for inquiry. If death occurs at a later period from disorganisation of the lungs with all the usual symptoms of phthisis, then our ordinary means of investigation are put into requisition to discover in what way the morbid appearances differ from those which are usually met with; whether the fibroid changes are peculiar, and whether softer deposits usually styled tuberculous should here receive a specific name. It cannot be maintained that we find anything more than thickening of the walls of the air-vesicles and a growth of imperfect fibre tissue mingled with nuclei, and these changes are not characteristic.

At the present time we must admit that there are true syphilitic gummata to be met with in the lungs; also that there are more chronic formations removable by remedies, which are probably of a fibro-plastic character, and which, if not removed, develop into true fibroid changes, and produce the phthisical lung; thirdly, there may be a more general and diffused hepatization of the lung due to the syphilitic influence, and this is a form occasionally met with in children.

The *hœmoptoic* variety of phthisis is one which has quite recently been added to the nomenclature, but not yet been accepted by all pathologists. It is a disease said to have its origin in an accidental coagulation of blood in the tissue of the lung. Hitherto the opinion of Laennec has been almost universally adopted, that hæmoptysis is a symptom of incipient tubercular disease, and even when no physical signs are apparent the disease has been assumed, since in so many cases a disorganisation of the lung has sooner or later followed. The more recent opinion is that an accidental hæmorrhage into the lung may itself lay the foundation for phthisis; that depositions of blood may set up a chronic form of pneumonia, and this undergoing the caseous degeneration may lead to the disorganisation of the lung; or even that the blood-clots themselves, by their presence and obliteration of the vessels, may undergo disintegration, and so give the starting-point for further changes. The subject of *phthisis ab hœmoptoë* is still under discussion, and requires much more illustration than it has yet received, the doctrine being by no means generally accepted. At the present time most pathologists would consider phthisis ab hæmoptoë to be due to tubercular bacilli aspirated with the blood into the alveoli of the lungs, rather than to the presence of ordinary blood in the pulmonary tissue.

A very good paper, with illustrations of hæmorrhagic relics and nodules, by Dr. Reginald Thompson, in the 'Med.-Chir. Trans.,' may be consulted.

Under the term phthisis are also included cases of disorganisation of the lungs induced by the entrance into the lung of irritant substances contained in the air, and thus we speak of *miners' phthisis* (*pneumonoconiosis anthracotica*, or *anthracosis*), *millstone grinders' phthisis* (*siderosis* and *chalicosis*), or what appears to be the same thing, *grinders' asthma.* The term phthisis is used because a disorganisation of the pulmonary tissue is apt to occur, but from the nature of the morbid process this class of disease might with equal propriety have been described under the name of chronic pneumonia. The subject has been fully investigated by Drs. Peacock and Greenhow, and they have found that particles of foreign matter, mostly mineral, are drawn in with the air into the tissue of the lung, lodge there, and set up a chronic inflammation accompanied by the usual exudations, and that these softening and disorganising destroy the lung after the manner of phthisis or a bronchial phthisis.

In the coal-miners' phthisis or *anthracosis* the lungs are black, a black juice can be squeezed out of them, the secretion from the tubes contains pigment in the form of granules or in cells, and pigment may be found in the alveoli and their walls, occupying also the lymphatics between the lobules which pass to the lymphatic glands, which are also blackened. A section of the lung shows pigment and inflammatory products surrounding the tubes, as well as siliceous matter and alumina. In a case of potters' asthma or phthisis or *aluminosis*, Dr. Greenhow found the same minerals in the lungs as were contained in the dry dust given off in the process of china scouring, and in a case of French millstone grinders' phthisis Dr. Peacock found siliceous and carboniferous particles, some of these being sharp and angular, corresponding to what was discovered in the dust of the workshop. In the Cornish miners' lung Dr. Peacock has been unable to find any foreign matters, and thus he regarded the disease to which they seemed liable as ordinary tubercular phthisis. In other cases the foreign matters by their irritation lead to the production of hard nodules through the lung, or if they soften, end in the destruction of the tissue.

Those who maintain the unity of phthisis would also include those cases as tubercular. The conditions of life of these workmen, they say, are exactly of that kind which would induce pulmonary disease. Moreover, the state of the lung itself containing fibrous and cheesy deposits would point to a special cause for its production.

**Pigment, or Spurious Melanosis.**—As a rule, the colouring matter in the lung increases in proportion to age, and thus a child's lung is comparatively white, while an old person's is of a blackish hue; consequently, all diseases at the two respective ages show a difference in colour; this is especially the case in chronic inflammation of the organ, where the older the person the darker will be the colour of the disease, the pigment having its origin in the hæmatin thrown out during the process of inflammation; sometimes the lung becomes excessively black where indurated. The most remarkable condition, however, connected with pigment is that called spurious melanosis, or, vulgarly, *black spit*, occurring in those who have breathed a carbonaceous atmosphere. The fact of a black lung being found in those who had breathed a black atmosphere at once led to the conclusion of a connection between them; but then the difficulty arose, how came it to pass? In the first place, let us inquire as to the nature of the pigment. In an ordinary lung we find it forming dark patches in the tissue; and we know that the secretion from the bronchial tubes contains pigment. We observe in the sputum some black specks, and if we place this under the microscope, we have a very striking object; we see the ordinary mucous globules, with epithelium, &c., and amongst these some larger cells of various sizes, containing numerous black particles, showing that in the secretion the colouring matter is, so to speak, organised—that is, it is contained in organic cells. In persons who work in coal-mines, and at trades where a carbonaceous atmosphere is respired, the expectoration is inordinately black; and in some cases these persons fall into a bad state of health, and die with disorganisation of the lungs, called in general terms, phthisis, which may be of the tubercular or chronic pneumonic character. Apart, however, from such disease and disorganisation, the lungs in such persons may become blackened, as seen in our specimens from miners of Newcastle; in these no change can be discovered beyond the whole tissue being quite black, as if the organ had been soaked in a black fluid. In a case which came before us not long ago of a man who had worked for many years in a gas factory, the lungs were indurated and very black, so that a quantity of dark juice came out when the part was scraped, and when placed on paper looked like Indian ink. In the same way the bronchial glands were enlarged, and converted into black masses, which could be squeezed into a paste composed almost entirely of pigment. In this lung there was considerably more than in an ordinary case of chronic pneumonia, since it stained the finger when touched. The pigmentary matter has been analysed by Christison, and found to consist of a hydro-carbon, and to be combustible like coal; and even said, on microscopic examination, to resemble coal dust; but the great difficulty is in ascertaining the direct connection between the carbon in the atmosphere and that in the lung. This difficulty in seeing how it can penetrate the tissue has caused some to think that the production of pigment was merely a secondary or accidental circumstance, seeing that it is a natural constituent of the lung, and is always produced wherever a chronic inflammation exists; and therefore that the irritating quality of vicious atmosphere breathed by the miners is merely instrumental in setting up this disease. Such explanation, however, is not satisfactory, for it is especially in those who breathe a black air that this is found; and moreover, as we have just now shown, a lung may be blackened without being diseased. Does the impurity, then, contained in the air actually pass through the air-passages into the lung tissue, and even to the bronchial glands? Such a supposition appears less improbable since experiment has shown that inorganic and insoluble substances like carbon, sulphur, or chalk, can pass through membranes, and may be taken up from the intestinal canal, and enter the lymphatics and mesenteric glands. Animals placed in a smoky atmosphere soon have their lungs blackened, so we must believe that the particles penetrate the lung to get to the interlobular tissue and go on to the lymphatics. It is thought that the black matter breathed by the miners is due to an impalpable form of carbon given off by their lamps, and not to the dust of coal; and in corroboration of this it may be mentioned, that at the present time there is a man in the hospital who is suffering from bronchitis, with one side of the chest dull, denoting probably chronic pneumonia, and the expectoration is very black. Now, this man has worked for years in the vaults of the London Docks, where torches and candles are continually blazing; and this is not the only instance of the kind which has come under our notice.

**Morbid Growths.**—These, for the most part, are secondary, and are of all kinds; probably there is no description of tumour occurring on the surface of the body that may not recur in the lungs, and thus we meet with cartilaginous, bony, myeloid, as well as more malignant growths; but very few of them occur as primary diseases of the lungs.

*Cancer and malignant disease*.—Primary cancer of the lung is a very rare disease, but when the organ with the neighbouring tissues has become involved in carcinoma to a great extent, the general term of *intrathoracic cancer* has been given. It may cause surprise, perhaps, to hear the opinion that primary cancer is uncommon, considering how frequently we meet with carcinoma of this organ; but the explanation is this, that in those cases where this constitutes the sole or principal disease, the morbid product has commenced in the bronchial glands or peribronchial tissue, and involved the lung secondarily. We would therefore say that cancer of the lung is generally of *two kinds*,—that in which the parenchyma is filled with cancerous masses, *secondary* to a similar affection in another part; and that in which the lung is occupied by a mass of disease, perhaps limited to the chest, and which, in contradistinction to the other, may be called *primary*, although it has its origin really in the bronchial tissues. The first form, where the cancer is found diffused through the lungs, is met with in cases where the disease has originated elsewhere, as after the removal of a cancer from a limb, or following the same disease of stomach, liver, or other abdominal organs; also, often after cancer of breast, either as distinct and separate deposits, or by actual contact from growth through the thoracic parietes. The cancer may be scattered as minute turbercles throughout the tissue, or in the form of large nodules, and very frequently seen as projecting flattened nodules on the surface of the lung. Sometimes, as before mentioned, they appear on the pleura, as hard, smooth plates, resembling cartilage.

The second form of cancer, intrathoracic or *primary*, as it is generally called, because, perhaps, the only part of the body affected, or from constituting the disease from which the patient suffers and dies, has spread, in most cases, from the bronchial or mediastinal glands, and then crept into the parenchyma along the outside and course of the tubes, but in what structure it has had its origin is not very clear, whether in the peribronchial tissue, bronchial glands, or connective tissue. Generally, a large mass may be found encircling the root of the lung and involving the organ at that part; if a section be made, the cancer will be found forming a layer around the tube, and proceeding with it, sometimes, to the most distant part of the lung; thus, if the organ be cut through, no disease may be found in the parenchyma, but the sections of the tubes will be seen encircled by a dense mass of white tissue. In some places, particularly if the disease be of the very hard or scirrhous kind, the tubes will be found contracted, and in some cases cancerous tubera will be discovered growing through the tubes into the interior. In some cases, where we find a large cancerous tumour in the chest, and it would appear most unlikely that the disease proceeded from the root of the organ, we may, on removing the parts, find the lung compressed and comparatively unaffected, while the adventitious growth has sprung up on one side, and really in the way named. In some of our specimens cancer is seen on the surface of lung. A *true primary* cancer of the lung is so rare that we have only one specimen to show. This has small nodules scattered throughout it, and none others observable in the body.

In most cases of cancer of the lung the pulmonary tissue is also involved in a destructive pneumonia. Portions of lung are hepatized and breaking down. This may arise from the irritation of the new growth, from plugging of the bronchial tubes, or from implication of the pulmonary nervous plexus.

It is not unusual also to find chronic pneumonic deposits, and then apparently a cancerous is associated with a phthisical disease. There is no doubt that tubercle and cancer may occur together in the same lung. We have seen a lung containing large phthisical cavities also the subject of cancer.

The form of secondary growth mostly met with is *medullary carcinoma*. It is vascular, giving out much milky juice; but seldom, however, very soft, unless it grows quite out from the surface. When the disease exists to a less amount it is often harder, contains a fibrous matrix, and may be styled *scirrhous;* an example of this we saw the other day in a woman who died of cancer of the breast. *Melanosis*, we have repeatedly before said, may be nothing but cancer accidentally coloured by pigment, as we think is the case in one example, where it returned in the lungs after removal of the eye.

The so-called primary tumours originate often in the lymphatic tissue at the root of the lung, and then extend into the parenchyma of the organ. In some cases, where the glandular growth is composed of a mass of cells contained in a fine stroma, the term lymphosarcoma would be more appropriate. Dr. Pye-Smith had a rare case of colloid disease of the lungs, both organs being full of translucent growths the size of peas. The disease was secondary to that of the stomach. We have one specimen of colloid disease which appeared to have its origin in the bronchial glands.

*Epithelioma.*—In cases of epithelial cancer of œsophagus we have seen, in one or two instances, the adjacent part of the lung involved in the disease, and in one case distinct deposits at a little distance. In one case where it was found in connection with the same disease in the larynx we had reason to believe that the germs had been sucked into the pulmonary tissue, and there propagated.

It may also be secondary to more distant disease, and we have seen well-marked capsulated epithelioma in connection with the same affection in the neck; also secondary to epithelial cancer of the tongue, the bladder, and the colon.

*Recurrent or malignant fibroid—Sarcoma.*—These tumours are generally secondary to the same disease elsewhere, as when, for example, a recurrent fibroid tumour has been repeatedly removed from an extremity, and the disease at last appears in the lungs. It is not very common, and may be called semi-malignant. In one specimen such tumours are seen which proved fatal in the lungs after similar ones had been removed from the skin several times; they consist of hard, round, firm fibrous masses. In another there are growths exactly resembling that in the uterus, which might be called *myosarcoma.* Another specimen shows a primary disease of this character, where the lung is involved in a fibrous or fibro-cellular growth; it is the only case of the kind we have seen. In a case regarded as primary sarcoma of the root of the lung there were secondary tumours in the liver and brain.

*Osteosarcoma.*—This disease is merely a fibroid tumour, with the addition of bone, owing to its proximity to osseous structure; sometimes, after removal, the same form of growth appears in the lungs, as round tumours composed of fibre and bone, the latter being true osseous structure, and constituting, generally, their circumference, as seen in several of our specimens.

*Osteoid cancer, malignant osteoid, or osteo-chondroma.*—This is a bony growth of a malignant nature, as we have already described, and it may occur secondarily in the lungs, after removal from another part.

Bone may, however, occur as a primary disease, although so rarely that it may be a question whether or not a malignant osseous tumour has not been present as a primary disease in some part of the body, and overlooked. We have already alluded to a case where bony plates were found on the surface of the lung.

*Cartilage, or enchondroma.*—This is not common in the lung, either as primary or secondary disease, but is occasionally met with under both forms; thus, in the remarkable specimen in St. Bartholomew's Hospital, described by Sir James Paget, where the lungs are full of cartilage, it was secondary to enchondroma of the testes, whilst the specimen of cartilage in our Museum removed from the lung purports to be a primary and local growth, and there is another where the disease began in the bronchial tubes and then penetrated the lungs as in cancer.

*Myeloid.*—Our Museum has a specimen showing a myeloid disease of the lung. This form of growth is probably much less malignant than any we have mentioned, and this constitutes the first specimen we have seen, the disease being generally quite local and innocent. We can see by the mode of growth how it differs from cancer; instead of being in the substance and infiltrating it, it springs from the surface, and thus we notice the pendulous character of these tumours; there is no other specimen of growth resembling it. It shows the true myeloid structure, consisting of a red-coloured soft material, and under the microscope characteristic giant-cells.

Various combinations of these growths may occur in the lungs, as in the original tumours, and we have found masses composed of cancer, myeloid, and bone, as in the primary growth which was removed from the leg.

*Tubercle.*—This we have already mentioned. Its favourite seat is the upper part of the lung. Much has been written about the seat of tubercle, and its frequency of occurrence in one organ rather than another. Louis has said that, after the age of fifteen, it can never occur in any organ without the lung being affected. He is probably right, and might even have carried the age still lower; for speaking in general terms, and allowing only a very few exceptions, tubercle may be said to be never or very seldom developed in the interior of the body without the lungs being affected. We have already said that, in acute hydrocephalus (and the same is true in most other tubercular affections), the lungs may nearly always be found to contain tubercles. The age mentioned by Louis is interesting, as being that of puberty: the time, certainly, in which tubercle is wont to show itself in these organs: indeed, we are convinced of the truth of the observation made several years ago by Dr. G. H. Barlow, that tubercle is developed in a particular organ according to its activity, and thus it is that the brain of infants is the most susceptible; at a later period the abdomen; and at the age of puberty, when the chest expands, the lungs are the most susceptible; and thus phthisis is comparatively rare before this period. Tubercles are very rarely congenital; only a few cases have been recorded, as two in infants three months old. In cases of *acute miliary tuberculosis*, where the whole of both lungs is stuffed with tubercles from apex to base, death occurs long before any disorganising process can ensue, and the affection is generally accompanied by a bronchitis, and the lung found red and very vascular.

*Hydatids.*—These may grow in the lungs as well as in other organs: they, however, are rare; for in the majority of cases, where they are expectorated, the seat of the parasite is the liver, it having made its way upward through the diaphragm into the lung. Sometimes death ensues, but very often the lung and liver coalesce, and recovery takes place. In one of our specimens a cyst may be seen making its way through from the liver, and in another the hydatids are apparently formed in the lung.

*Actinomycosis* has been observed in the lung, the disease beginning apparently in the bronchial tubes, and then attacking the lung and ending in abscess.

*Bronchial phthisis.*—This is a tuberculous disease of the bronchial glands which generally involves the lung, so that it is only when pulmonary symptoms have arisen that the child comes under medical notice. A history of measles is so common in such cases that it may be supposed, in connection with the accompanying bronchitis, some inflammation and enlargement of the bronchial glands take place; this is followed by caseation and softening of the inflammatory product. A large mass of bronchial glands may be thus affected, and, in course of time, the neighbouring pulmonary tissue becomes implicated in the same form of disease. Death may come about by a general destruction of the lung, and not infrequently the bronchial disease may set up a pleurisy or empyema. We have seen the ulcerative process penetrate the œsophagus, and the escape of the contents of this tube set up a pleurisy. We have seen a case also end by a fatal hæmorrhage, and our Museum contains a bronchial gland which became free, and fell into the bronchus, and suffocated the child.

# DISEASES OF THE ALIMENTARY CANAL

## TONGUE

**Malformation.**—In extreme malformation of the oral cavity in unviable abortions the tongue may be absent or only represented by a stump at the back of the cavity. The tongue may be congenitally unnaturally fixed by adhesion to the floor of the mouth, or its frænum may extend too far towards the tip or be too short.

**Hypertrophy.**—The tongue also is subject to a very curious hypertrophy—*Macroglossia*—which is often congenital, but may increase after birth and give rise to great suffering, requiring partial removal, and, perhaps, causing death by inanition or suffocation. It generally affects the anterior part of the organ. The tongue is found greatly swollen, it hangs out of and stretches the mouth. Virchow first showed the nature of the disease : the enlargement is caused chiefly by dilatation of the lymphatic spaces in the organ, so that it becomes like a sponge, with channels full of a clear fluid. The muscle is generally not increased, but there may be irritative increase of the connective tissue and thickening of the epithelium. The whole condition is like that of the legs in elephantiasis. In a few cases the tongue has been similarly enlarged by greatly-developed *nævus.* This sometimes degenerates into a blood tumour, but true angiomata are rare.

**Atrophy** of the tongue depends usually upon affection of the motor nerves. Thus it has been observed to waste in bulbar paralysis, and sometimes in tabes. In a case related by Sir James Paget, there was unilateral atrophy in consequence of disease at the base of the cranium, which involved the ninth nerve.

**Fur.**—This is generally found in connection with febrile complaints. It may be universal or limited to one part ; the secretion may be white, yellow, brown, or black ; it may be soft and creamy or tenacious and viscid. The mucous membrane at the same time may be universally injected and of a deep red colour, or the papillæ highly vascular and projecting. It may be also cracked, bleeding, or ulcerated. The partial fur on the tongue had been alluded to by Mr. Hilton as due to a nutritive cause from some implication of the nerve, but Mr. Hutchinson explains the circumstance in a more simple way by supposing that the patient does not masticate on one side of his mouth, and the fur fails therefore to be rubbed off the tongue.

**Inflammation.**—We may meet with general inflammation, or *glossitis*, as a part of a diffused inflammation of the mouth, through injuries, or salivation, or smallpox, &c.; but sometimes we find such glossitis as an isolated affection. An example of this we may see in a specimen of an immense enlargement of the tongue, which was the cause of a woman's death in the course of a day or two. We have found pyæmic abscesses in the tongue. In more chronic cases, an effusion occurs gradually into the substance of the tongue, and its swelling may remain for a considerable time. *Ulceration* of a simple or aphthous character is also very common on the tongue.

**Chronic Glossitis.**—The papillæ of the tongue are often enlarged, the circumvallate being hypertrophied so as to resemble small tumours, or the filiform changed into long threads like hairs.

Sometimes the epithelium is thickened into hard scaly plates, and to this condition the names of *psoriasis*, *ichthyosis*, or *tylosis linguæ*, are given. Mr. Hulke, many years ago, described the affection as consisting of raised white patches on the surface of the tongue. These sometimes occupy a large space, and are mostly found on the dorsum of the tongue, so that the filiform papillæ are mostly affected. The raised white patches can be felt thickened by passing the finger over them. There is considerable hypertrophy of the epithelial and papillary elements, whilst the mucous membrane beneath is healthy as well as the muscular substance. In this way they differ both from epithelioma and syphilis. They have been known, however, to pass into epithelial cancer.

**A superficial Glossitis or smooth Tongue** has been described by Mr. Butlin. It is the case where there is a thickening of the corium with cell infiltration and a thickening of the epidermis, but the latter is devoid of papillæ and is as smooth as the cornea. It differs from ichthyosis in being red, smooth, and glossy; but Mr. Butlin suggests, nevertheless, that it may be a variety or different stage of the same disease. There are often ulcerations or cracks around. The disease has been called *leukoplakia buccalis.*

**Tubercle** may occur as an isolated deposit. This may soften until an ulcer is formed. Several cases of tuberculous ulcer of the tongue have been observed in connection with phthisis.

**Syphilitic** disease occurs at a comparatively late stage in the malady, being preceded by mucous patches on the throat. Similar ones may then be observed on the tongue, to be followed by small superficial gummata in the corium. The most characteristic form is that where a chronic hypertrophic inflammation occurs in the muscular substance producing a fibro-cellular or gummatous material. Diffused lumps are thus formed which may ulcerate, causing indurated sores with hard edges, and which have often been mistaken for cancer.

*Morbid growths.*—Although most of the knots in the tongue are syphilitic, yet new growths are not uncommonly met with. *Cancer* is by far the most frequent, and it is most commonly of the epithelial variety; it mostly does not spread beyond the contiguous parts and adjacent lymphatic glands. This form of disease is a striking object for the microscope in the aspect of the large single or mother-cells amongst the muscular fibrillæ. It puts on various forms, and may begin as a hardish knob, which after a time ulcerates, but as a rule it commences as a fissure or ulcer, which soon becomes ragged; this ulcer often arising in the first instance from the irritation of a tooth. Cancer of the tongue is by far more common in men than in women.

Curious congenital *pendulous* tumours are sometimes met with on the tongue; they have a structure like that of nasal polypi. *Cysticerci* are sometimes found in the tongue, also nævi. *Sebaceous* or *dermoid cysts* are occasionally removed by the surgeon. They are probably congenital.

## MOUTH AND FAUCES

**Malformation.**—The mouth rather frequently shows malformations. We need scarcely consider its conditions in unviable fœtuses, in which it may be continuous with the nasal fossæ and even with the orbits, or the lower jaw may be absent. There are many faults of its structure compatible with life. Thus the mouth may be made small or closed by cohesion of the lips, or these may be ill-formed and imperfectly distinguished, the orifice being stiff and small on that account; or a fissure may extend the mouth on one or both sides nearly to the ears. But the most common and important malformation is that known as *hare-lip.* In this condition a fissure, or two symmetrical fissures, are found in the upper lip corresponding in position with the junction of the fœtal intermaxillary with the superior maxillary bone. The fissures may extend here into the jaw through non-union of these bones, and if the case be severe the portions included between the fissures may be absent, or much reduced in size, causing a great gap in the upper lip. The *uvula* may be *bifid,* as seen in one of the specimens, or the *soft palate* may be "*cleft*" through non-union of its halves, or the cleft may implicate the *hard palate,* when it may rarely be seen on both sides of the vomer, or else be single; this condition may complicate hare-lip. The soft palate may be absent. Writers on "monsters" give a list of technical names for the various malformations affecting the mouth and jaws.

**Inflammation.**—We will first speak of the affections of the back part of the throat, diseases in which the pharynx is also commonly involved, for these parts are intimately connected in their pathology. One of the commonest diseases is simple sore throat; if this be severe it receives the name of *cynanche tonsillaris*, inasmuch as the violence of the inflammation is spent on the tonsils, so that they swell greatly through œdema and increase of their lymph-gland-like structure; if yet more severe, the inflammation often ends in suppuration, producing tonsillar abscess or *quinsy*. A sore throat may arise, also, from other causes, as *scarlatina;* in this form great swelling results, which does not terminate in abscess, but rather in sloughing if the case be grave, although a superficial suppuration or ulceration not infrequently happens. The structure of the scarlatinous tonsil is not microscopically to be distinguished from that of the swollen Peyer's patches of enterica. In ordinary cases the inflammation has a tendency to affect the Eustachian tube and internal ear. A slight sore throat occurs, also, frequently in the exanthemata, fevers, erysipelas, &c. We have seen suppurating tonsils and glands at the root of the tongue in rheumatic fever. Virchow describes a great enlargement of the tonsils and glands at the root of the tongue in hydrophobia; this he calls *Lyssic angina.*

Besides the superficial or catarrhal inflammation of the throat there is a very important chronic inflammation known as *granular pharyngitis.* It must be remembered that the epithelium on the posterior part of the throat is cylindrical, that there are no papillæ, and that, besides the simple follicles, there are compound or racemose glands, which can be seen by the naked eye. In the affection of which we are now speaking these glands become closed and much enlarged, so as to give the pharynx a granular appearance. The centre of these granulations corresponds to the ducts, and surrounding them is a white layer of epithelial cells. The mucous membrane has thus often an irregular surface with minute ulcerations, whilst it is at the same time very vascular.

Sometimes the gland contents grow to that extent that the term *adenoid vegetations* has to be given to the disease. These constitute distinct masses of glandular growth to be seen on the pharynx, and extending often to the back of the nares. Dr. Goodhart has paid much attention to the subject, and given a good description of these vegetations, which deserve sometimes the name of polypi, as well as the accompanying hypertrophy of the mucous membrane. In the case of an infant he found the whole mucous membrane of the pharynx extremely thickened.

After repeated attacks of cynanche the *tonsils* become permanently *enlarged*, constituting a very common and troublesome affection. We may see this in the youngest infants, so that it appears, sometimes, to be congenital; in older persons, these glands are readily excised, and numerous specimens may be seen on our shelves; they almost constitute tumours; when large they are chiefly composed of lymphoid structure like that natural to the tonsillar follicles, but sometimes they consist of tough fibre tissue, developed in the substance of the gland. Their surface presents this peculiar cribriform appearance from enlargement of the mouths of the crypts around which the follicles of the glands are situated. Occasionally, chalky concretions form in the tonsils, and these are composed of phosphate of lime. Sometimes, also, the whole soft palate becomes thickened and indurated. We often find the tonsils replaced by the scars of old ulcers.

Suppuration may be found not only in the tonsil, but at the back of the pharynx, in a *phlegmonous* form; it may then have a deep-seated cause, as disease of the bone; for caries of the cervical vertebræ very frequently causes a retro-pharyngeal abscess, as we have already mentioned. But in young children retro-pharyngeal abscess may occur as a primary affection, or as a consequence of scarlatina, or some other fever. The abscess forms a swelling at the back of the pharynx, pressing forwards on the glottis so as to endanger suffocation. We have seen large abscesses arise here from the irritation of ill-placed wisdom teeth, and also in states of exhaustion without any such local cause.

*Diphtheria.*—This is now recognised as a distinct specific affection. The local peculiarity of the disease is the formation of a membrane, hence the name given to it; and, in consequence, the term diphtheritic has been, of late years, used very generally for any inflammation attended by a membranous exudation like that of croup, whether seen in the throat, intestine, eye, or in other parts. In various diseases accompanied by sore throat, a considerable extent of pellicular exudation may occur; but diphtheria appears to be altogether peculiar: in it a membranous pellicle is formed, which may be of any degree of consistence; when pretty firm it sometimes can be removed in one piece, and then represents a perfect cast of the part of the mouth or throat, leaving the surface beneath livid and congested, or, perhaps, ulcerated; actual sloughing is rare. The disease begins, first of all, by a redness on the throat, and soon white patches are seen here and there, until they coalesce into one membrane, covering the pharynx and posterior nares, and extending into the trachea and bronchi. But topical varieties are met with frequently, wherein the disease fastens primarily on only one of these parts and extends but little to the rest. The corresponding lymphatic glands are enlarged considerably. Some uncertainty prevails as to the character of the pellicle, some being of opinion that it is a mere exudation, such as is often seen on an inflamed mucous surface, while others consider that it is due to a parasite.

The membrane can be usually raised and detached, and when examined microscopically is seen to consist of layers of fibrin in which are intermixed lymph-corpuscles, some blood-corpuscles, and micrococci. The false membrane is in close contact with the corium whose papillæ penetrate it, and the uvula and soft palate are also swollen from infiltration of inflammatory products. The subject has already been considered under "Croup."

*Specific ulceration.*—The various forms of sore throats may go on to ulceration: cynanche often leading to small simple ulcers in the tonsils.

*Tuberculous ulcers* of the throat, as a rule, proceed from the larynx, and thus are unlike the syphilitic, which may attack the pharynx or larynx independently. In the former, we find the disease has commenced in the larynx in connection with phthisis, and then has spread over the glottis into the pharynx and throat. Occasionally, however, tubercles may be found isolated on the palate, tonsils, and other parts of the mouth, commencing as white spots, and then softening until ulcers are formed. The *syphilitic ulcer* is much more common. We will just mention the occasional occurrence of primary chancre in the mouth, but syphilitic ulceration is generally of two kinds:—First, we meet with ulcers during the eruptive or so-called "secondary" stage; these are generally callous, well defined, rather excavated ulcers with a greyish floor, they are commonly symmetrical and may appear in any part of the mouth, though they generally prefer the tonsils. The second kind are in the "tertiary" stage, perhaps long after the syphilitic fever is over. These are unsymmetrical ulcers and may spread widely and deeply, so that in cicatrising they may lead to serious stricture, or they may destroy life by their extent. We met a case where the left half of the hyoid bone stuck out of such an ulcer. These may affect the larynx low down, or destroy the soft palate and creep upwards into the posterior nares, or involve also the roof of the mouth. The first form may begin as a mucous patch, which may soften and lead to a superficial ulcer; the latter may begin as a gumma deeper seated, which may soften and lead to an ulcer, accompanied by great thickening around it. Congenital syphilitic ulcers may also be met with, as in the case of a puny lad whose soft palate was entirely destroyed by ulceration, which rapidly healed under the use of iodide of potassium.

*Stomatitis vesicularis, or aphthæ.*—This is a well-known affection, occurring in children, and in adults towards the close of many diseases. A white pellicle appears, at first in small patches, and extends until it covers the tongue, mouth, and fauces. If the pellicle be examined, it will be often found to be composed of the mycelium and sporules of a parasitic fungus called *oïdium albicans.* It is not, however, quite clear how far such a parasite is a mere accident of the disease, or an essential part, but transplantation of the fungus has produced thrush in healthy children, so that there is no doubt it plays an important part in causing the disease, though a weakly state of the child and an acid state of the oral secretions are also important elements in its production; hence attention must not be fixed on the local condition only. Indeed, there is evidence that the fungus is not always present in these pellicular diseases, being absent in some very bad cases of throat affection. Aphthæ may extend into the pharynx and œsophagus. We must remember that an analogous form of growth is found between the teeth of those who do not habitually use the toothbrush, but who are in perfect health. To this last parasite the name *Leptothrix buccalis* has been given by Robin. Many other forms of bacteria are described as occurring in the mouth.

*Stomatitis follicularis* is generally of little importance; it appears in one or several swollen glands that may form ulcers, round and well defined; in the fauces they may be attended with a few days' feverishness in delicate children, and create an unnecessary alarm of diphtheria. We have once known such follicular ulceration extending throughout the cheeks and lips as a very obstinate disease in an adult.

*Stomatitis ulcerativa.*—This is a very common form of disease, and one which we see constantly among the out-patients, where ulcers may be found in various parts of the mouth, but especially along the gums.

*Pustular inflammation.*—We find, as a rule, in severe cases of smallpox, that the throat is covered with pustules.

We meet sometimes with *acute œdema* of the pharynx in Bright's disease. This is very dangerous from its tendency to involve the larynx.

*Stomatitis gangrenosa, cancrum oris, or noma.*—This disease is one we occasionally see in the hospital; it affects children, most frequently from three to five years of age, and is generally fatal. It occurs mostly after some fever, as measles, typhoid, &c., making its appearance as a slight swelling and pale red patch on the cheek; this rapidly becomes deep red, then decays and turns black. The disease, however, has meantime made a greater progress on the mucous surface, which exhibits a slough while yet the skin is red. The sloughing goes on rapidly within the mouth, so that the whole inside of the cheek becomes a foul grey and most fetid pulp, the opposite gums become invaded, and the teeth loosen at last. The cheek breaks down, and the tongue and pharynx are exposed, while the teeth fall out. In typical cases the disease commences thus in the substance of the cheek, and its mucous surface is first affected. But sometimes gangrene starts from the gums. Trousseau describes gangrene as arising from these, and reaching the cheeks after "diphtheria" has been several months in the gums. Yet this disease is quite different from ulcerative stomatitis. It is attended by severe constitutional disturbances, and perhaps delirium, but not by much suffering. It may be associated with similar disease of the genitalia. Recovery rarely occurs; but if so, it is generally with exfoliation of parts of the jaw. We have examined several cases of the disease after death, but have failed to find any good explanation of the affection, either locally or constitutionally. The blood-vessels of the face proceeding to the part have been carefully dissected, but nothing in them has been discovered to suggest a reason why even in great constitutional debility this part of the body should be especially affected ; and, as regards any general cause, the same difficulty exists, since sometimes no organic disease can be found, although in two or three cases a fatty liver has been met with. Lingard believes he has found special organisms in cancrum oris, and from his experiments thinks it is of the same nature as some diseases of the lower animals.

**Morbid Growths.**—We sometimes meet with hypertrophy of the submucous glands forming *adenoma:* these may reach a large size, and cysts may form in connection with them. Such adenoma has occurred also on the tongue congenitally. We have several specimens of *warty* growths, or *papillomata,* from the soft palate and uvula. They are more frequent on the lips. A drawing of one shows loops of blood-vessels covered with squamous epithelium; indeed, they resemble papillar or warty growths elsewhere. Sometimes a growth on the palate is flatter, harder, and more fibrous, so as to be called a *condyloma,* as seen in our specimen.

*Polypi* are mostly found growing in the pharynx and upper part of the œsophagus. These have the same structure as nasal polypi.

*Epulis.*—Using the term, like Virchow does, as an equivalent for tumours of the gums (which really come from the periosteum of the jaw) we shall find that many of these are of the nature of *sarcoma,* but especially of that variety in which Lebert, while framing his description of fibro-plastic tumours, first figured the cells which were called *myeloid,* but are now known as *giant cells.* In such kinds of epulis the cells may reach a size so great that they are visible to the naked eye. These cells are now known not to be peculiar to any kind of tumour; nevertheless, the great size and clear definition of them in these tumours of the jaw give such tumours a special interest. But an epulis may be simply a fibroma, or may be compounded of fibroma and sarcoma; it may be observed to recur very often after removal. It is very firm, springs from the periosteum, and is covered by gum tissue ; and one specimen has a few plates of bone in it, a condition not unfrequently met with. In another specimen, which came from a man who was said to have epulis, the tissue was *epithelial cancer* growing from the gum ; it rapidly returned after removal. It is an old question whether such cancer should be called epulis. This is a question of words, yet epithelial cancer is so well recognised, and its tendencies so well known, that it is always advisable to give the name when it is seen to be applicable. Probably most surgeons apply the term epulis to non-malignant growths.

This form of disease, *epithelioma,* we know, is very common on the lower lip, and its structure is seen then to great perfection.

*Sarcoma* and *carcinoma* occasionally attack the soft palate and tonsils, as in a woman lately in Mary Ward. We have met with *lymphomatous* cancer of the tonsils along with the same disease in the spleen, &c.

Dr. Taylor had a remarkable case where a large *lipomatous* tumour grew from the back of the pharynx of a child.

We have already shown a *horny* growth from the lip. *Angioma* or *nævus* is frequent there. We need not, indeed, recount all the diseases which the skin of the lips suffers, like other parts of the tegument. *Lupus* may extend within the lips and reach the tongue or throat.

We might here allude also to another morbid state, which it is well to look for sometimes on the dead subject as well as the living; we mean the *lead line* on the gums; this we recognise as a blue or dark slate-coloured rim along their edges, as seen in our drawing. If examined more closely with a lens, we perceive the colouring matter has a dotted appearance from the papillæ being first affected. It is due to sulphide of lead formed from the sulphur of the mouth. In Addison's disease pigment may be found on the cheek and tongue.

*Ranula.*—This name is given, much in the same loose worn-out way as that of epulis on the gums, to cysts in the floor of the mouth under the tongue; cysts which have a bluish colour, may grow to a large size, and are full of viscid fluid. They are sometimes due to dilatation of the duct of the submaxillary or sublingual gland, and a specimen in our Museum shows very well the connection of a large ranula with Wharton's duct. At other times Wharton's duct has been found still pervious and discharging saliva, and then Rivini's ducts are probably dilated and form the cysts. Mr. Birkett has well portrayed the two forms in some drawings: those arising in the salivary ducts and those arising in distended mucous follicles.* Phosphatic calculi are sometimes found in ranulæ, as also in similar dilatations of the pancreatic duct.

*Dermoid cysts* in the mouth we have met with, one full of cholesterine and epithelium. One described by Mr. Waren Tay as containing fat, like butter, was probably of the same kind, and Dr. Hale White removed one from a living child which contained hair. Some examples of large *lipoma* have been found here and mistaken for ranula.

The **Salivary Glands** are liable to acute inflammation in mumps. The morbid anatomy of this disease is not very pronounced; the glands soon recover their natural state. We sometimes meet with suppurative inflammation of the salivary glands, as occasionally after scarlet or enteric fever, in diseases of the abdominal viscera. The microscope shows the suppuration to commence in the interstitial connective tissue rather than the glandular acini. We have a specimen of calculus from the submaxillary gland, the obstruction of the duct of which, as we said, is one cause of ranula. From the same cause in Steno's duct we may have salivary fistula. Amongst the new growths affecting these glands is the *fibro-enchondroma of the parotid*, and more rarely of the submaxillary. We do not think that it is clearly made out where such disease first begins, although the glands appear more or less involved. It is rare to find simple enchondroma, this being commonly mixed with fibroma or myxoma. *Cancer* of the salivary glands is sometimes met with; and occasionally with adenoid tissue.

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* 'Guy's Hospital Reports,' series 3, vol. v.

Mr. Jacobson, who has been working at these tumours, is inclined to Cohnheim's opinion that they have their origin in the relics of embryonic tissue, as there seems no other reason for their growth in this region. It is thought that some undeveloped cells remain during the differentiation of the branchial arches, and these may become simple tumours or develop into cysts or supernumerary auricles. In a mixed tumour, as with sarcoma, the origin would be in the connective tissue.

## PHARYNX AND ŒSOPHAGUS

The affections of the fauces we have already spoken of, and merely place the pharynx with the œsophagus so as to include the diseases of the upper part of this tube. We must refer back for the special diseases of the pharynx.

**Malformation.**—In monsters the digestive canal may be variously altered, but in otherwise normal children deviations have been found, as the œsophagus and aorta transposed, a case of which we shall find in the 'Guy's Hospital Reports.' Occasionally the œsophagus is found terminating in a *cul-de-sac*, or even opening into the trachea. We have met with pouches from the side of the pharynx behind the tonsil; such pouches have been described, as well as fine fistulous openings from the pharynx to the surface, representing a persistence of the fœtal branchial fissures.

On the cutaneous surface these may be met with between the jaw and sternum as small fistulous openings, secreting a thin mucus, and generally having a blind end; sometimes, however, opening into the pharynx or trachea.

Mr. Bland Sutton believes that the cysts so often found in the neck have their origin in the remains of the branchial arches. They have been described by many authors as *congenital cysts in the neck.* Mr. Treves exhibited one specimen at the Pathological Society which was as large as the child's head. Mr. Tom Smith has described them in 'St. Bartholomew's Reports,' as also Mr. Birkett in the 'Transactions' of the Royal Medical and Chirurgical Society. They were designated *hydrocele of the neck*, *serocystic disease*, and *hygromata.* They are sometimes of a dermoid character.

**Dilatation.**—Cases are recorded where the œsophagus has been dilated so as to form a kind of second stomach, and with this even a process of rumination has existed. In the Museum of St. Thomas's there is a remarkable case of dilatation of the whole of the œsophagus. It was found accidentally after death, and unconnected with any symptoms. A similar case of yet greater dilatation of the whole œsophagus is in our Museum.

Such specimens show a simple stricture of the lower end of the œsophagus, with enormous distension of the tube above. Both of our cases came from old persons who nearly all their lives had symptoms of obstruction. The Catalogue gives full particulars.

**Contraction or Stricture** is a form of disease very commonly spoken of, as if the œsophagus could be strictured in the same way as the urethra. This, however, is exceedingly rare, for in the vast majority of cases where the obstruction exists it is due to disease within the tube, or to some tumour pressing from without; in short, generally either a cancer of the œsophagus or aneurysm of the aorta. As, however, it is often certain that in the living subject such severe affections do not exist, the term stricture must still be conventionally used in those cases where there is a mere difficulty of swallowing; remembering that it is exceedingly rare to demonstrate such a condition, for during several years' observation in the post-mortem room we have never yet met with an instance of it; but we have found the muscle of the œsophagus thrice its normal thickness in heart disease, as if hypertrophied from obstruction by the pressure of the large heart. We have in the Museum one or two specimens which show that an ulcerative process may give rise to a cicatrisation and subsequent contraction. In one the constriction is at the lower part of the pharynx where it joins the œsophagus; it is no doubt a contracted ulcer, and may be syphilitic; in another the ulcer is of a doubtful character. The very remarkable specimen of the œsophagus from a boy shows great thickness of the walls at its lower part; this has caused much difficulty of deglutition, and was probably due to swallowing some irritant fluid long before. Cancer we shall presently speak of.

**Wounds** of the pharynx and œsophagus may heal, as seen in cut throat, or as in the cases of pharyngotomy we saw performed by Mr. Cock.

**Inflammation.**—*Idiopathic.*—An independent œsophagitis is probably a rare form of disease, at least it is one which is not often appreciable on the post-mortem table. We have met with small submucous abscesses apparently unimportant; but there can be no doubt that the œsophagus is sometimes affected in catarrh, as indicated by symptoms of gastric disturbance as well as difficulty of swallowing, and some cases of intense pain in swallowing have been called œsophagitis. We have already described the inflammations of the fauces; any of these, if severe, may extend down the œsophagus.

Pellicular inflammation of the pharynx and œsophagus is thus sometimes met with. It may be a true *diphtheritic* inflammation proceeding from the pharynx into the œsophagus; this, however, we believe is not frequent, though we have twice observed it, but *aphthæ* or *thrush*, the muguet of the French, we have several times observed to pass down the tube. A similar pellicular inflammation extending all down the œsophagus we have twice seen result from drinking boiling water; once there was pus in the mucous membrane and no pellicular layer. In conditions of extreme debility, *e. g.* advanced phthisis, prolonged low fever, &c., we have occasionally met with a circumscribed slough on the pharyngeal surface of the larynx (laryngeal part of the pharynx). But in children who have drunk boiling water, if life has been preserved for a day or two, we generally find only some lymph on the surface, and at the same time the mucous membrane abraded and shreddy in parts. If death have occurred rapidly, all we find is serous exudation into the submucous coat, whereby the walls of the tube are thicker and softer than usual, showing the first stage of inflammation. After the swallowing of poisons the mucous membrane may become indurated or destroyed. In some cases, as by sulphuric acid, the membrane is converted into a dark brown or thick substance; if the fluid has passed down rapidly, the rugæ only are affected, and the membrane may have been destroyed and loosened in patches.

*Ulcer.*—We have twice met with round ulcers in the œsophagus corresponding in character with the *simple* ulcer of the stomach, and in one case associated with such a simple ulcer. We also once saw in a *syphilitic* subject two yellowish gummatous patches in the œsophagus, and once saw the tube opened by a large soft syphilitic deposit outside it. And more rarely a *tubercular* ulcer may be met with. On our shelves is a specimen from a phthisical child, where a minute ulceration exists on the mucous membrane corresponding to the ulcers on the larynx and elsewhere.

Occasionally dilated veins are met with at the cardiac end of the œsophagus in consequence of portal congestion, and so produce "piles" here as in the more commonly affected rectum. And there are one or two cases on record where an ulcer occurred over one of these vascular patches and led to fatal hæmatemesis.

There will be seen on our shelves two or three specimens showing an *opening between the œsophagus and trachea*, the remarkable feature of which is that the aperture is circumscribed, and has no adventitious matter around it to indicate its nature; the disease not appearing cancerous, nor is there evidence of suppuration, no more being seen than a simple ulcerative process.

*Perforation of the œsophagus from without* is not very infrequent; we meet occasionally with the relics of bronchial glands attached to the œsophagus at the seats of old scars in its wall; thus showing bursting of old bronchial abscess into the œsophagus in childhood. We have seen such perforation cause pleurisy and empyema. Sometimes portions of necrotic vertebræ have come up from abscesses when spinal caries has opened into the œsophagus. Gangrenous cavities in the lungs or mediastinal abscesses may communicate with it, or aneurysms of the aorta burst into it.

**Morbid Growths.**—The most common is *cancer*. The harder *scirrhous* form of the disease is rare, but a specimen of stricture, with much thickening of the walls, appears to constitute an example of the disease. A less rare form, although not very common, is the *medullary* as in a specimen, where we see a large encephaloid tumour growing in the œsophagus. This generally affects the lower end and may create extensive secondary cancers in the viscera. Two cases that we examined were *adenoid* in their structure, one was soft carcinoma *reticulare*. The ordinary form of cancer, however, is the *epithelial*, and this is very common. In the upper part or pharynx it may appear as a distinct circumscribed growth or tumour growing from one side, generally the anterior wall. In most cases, however, in the upper part, and always when it occurs lower down, it involves the whole circumference of the tube, and then softening down produces a large cancerous ulcer. Occasionally a great extent of the mucous surface may be affected, and the disease reaches quite into the pharynx, where the growth has a warty appearance, and sometimes nearly the whole length of the tube is occupied by the disease in its sloughing stage. Sometimes the pharynx alone is destroyed by *cancerous* ulceration. A very frequent position for cancer is nearly opposite the bifurcation of the trachea, which is frequently involved. The extension of epithelial cancer is quite local, the œsophagus and neighbouring lymphatic glands being the only parts affected, except where the disease involves parts around in contiguity with it. Thus, the cancer may reach to the lung, and then, breaches of its continuity occurring either through lodgment of food and sloughing or through perforation by a probang, matters escape into the lung and form a foul, sloughy, pneumonic abscess. This occurs rather often and should be suspected if pneumonic symptoms arise. As a rule, we find that the destructive process advances more on the anterior than on the posterior side, for while the tube is rarely torn open on removing it from the spine, we find the front wall quite destroyed, and the disease invading the windpipe, lungs, and other parts; this position of the cancer may be accidental, owing to the less pressure in front during its growth. The surface is found sloughing, of a dark greenish colour, and very fetid; sometimes portions of dead tissue are lying loose upon it. On making a section quite through it, the adventitious tissue is seen beneath, and forming, sometimes, the sole continuation of the tube between the healthy parts. The extent of the disease is generally about three or four inches; it pervades the neighbouring glands, which are enlarged by the cancer; it often involves the pneumogastric nerves, and occasionally, the pericardium is entered but sometimes we find only a small cancerous nodule, placed so as to obstruct the tube and cause death. Extensive cancerous ulcerations in the upper part of the canal sometimes are fatal by hæmorrhage, through perforation of the carotid; or the aorta may be opened by cancer lower down, &c.

We have twice met with general suppuration in the tissues of the neck through perforation of the pharynx or œsophagus by cancer. Although so liable to primary cancer the œsophagus may be found passing unaffected close by cancer of the lung. Tumours of the muscular tissue or *myomata* are occasionally met with in the œsophagus. A specimen of Dr Fagge's shows a growth two inches long, and made up of interlacing smooth muscular fibres. Polypi, such as are found in the nose or uterus, are occasionally seen there.

*Warty growths on œsophagus.* — The mucous membrane of the œsophagus is provided with papillæ, and sometimes these are increased so as to form warts, resembling warts on other parts, but more usually they consist of white flat patches, slightly raised above the surface of the mucous membrane. They consist chiefly of an increased growth of epithelium, and thus resemble the corns on the inside of the hand. They are very frequent in cases of heart disease, especially in elderly people. The Museum of Comparative Anatomy contains a remarkable specimen of the gullet of an ox of which the whole surface is covered with a shaggy or cauliflower growth.

*Cysts* in the œsophagus are met with occasionally, as seen in one of our specimens; this did not produce any symptoms. They are formed in the walls from the mucous follicles, and contain a thick mucoid fluid.

**Digestive Solution.**—We shall presently speak of specimens of stomach dissolved by the gastric juice; and the same sometimes occurs in the œsophagus, when the contents may escape into the pleural cavity, as we have already mentioned. We always find the contents to be highly acid under these circumstances, and the soft dissolved appearance of the part is quite characteristic; there is no sign of inflammatory reaction around, as in ulcer. Amongst our specimens there is an œsophagus partially dissolved by the gastric juice; and another in a less degree where merely the longitudinal rugæ are removed or hanging in shreds. While the body is supine, the gastric contents may pass upwards into the œsophagus; and this is important to remember; for if these contents find their way into the pharynx, and then down the trachea into the bronchi, as often occurs, we might be somewhat puzzled to find such unusual matters in the lungs; the strong acidity of the contents of the trachea under these circumstances will always guide us.

In connection with digestive solution is the question of *spontaneous laceration.* Two or three cases have been taken to the Pathological Society, where after violent vomiting a rent was found in the œsophagus. No decision was arrived at, as to whether the perforation had occurred before or after death.

**Foreign Bodies.**—Large masses of food may be found impacted in the pharynx or œsophagus. A large piece of meat was found in the pharynx of a man who was brought in dead, suffocated by it, though it appeared within easy reach of one's fingers. Generally there is either extreme youth or softening of the pons, &c., to explain the accident by carelessness, or paralysis of deglutition. In an œsophagus in the Museum may be seen a large piece of pudding impacted. It killed the child, who was dead when brought to the hospital. There is another with a muslin handkerchief tightly stuffed in the pharynx, due probably to a suicidal act. There is also a specimen of a bird's heart which blocked the œsophagus for some hours. Of late we have heard of several instances where death has been caused by the impaction of false teeth in the œsophagus. Sometimes fish bones, or pieces of bone, carelessly swallowed with meat are lodged in the œsophagus and may then ulcerate their way through, sometimes into the aorta, causing death by hæmorrhage.

## PERITONEUM

**Inflammation.**—*Acute.*—This is characterised by the same appearance met with in other serous membranes already described, which, however, are seen in greater perfection in the peritoneum. Thus, after a few hours of inflammation, the membrane will be found reddened from hyperæmia, while, at the same time, the intestines are distended, their muscles having lost contractile power, producing the tympanites observed during life. At a later period an exudation of lymph will be poured out upon it. This is often excessive, and is seen in large flakes covering all parts of the surface. In the earliest period, however, we must look carefully for the lesser signs of inflammation; thus the exudation may be so slight that it is only by scraping with a scalpel that the lymph becomes perceptible, though the dull look of the surface and its greasy feel are sufficient to detect it. The redness of the membrane is then of importance; it will be found in bands along the intestines, which bands represent the spaces where the cylindrical forms of the intestines tend to leave intervals between the coils, so as to favour the lodgment of blood there by a sort of suction; on these suction bands, also, the exudation is first thrown out, while the more closely pressed surfaces have little lymph between, and very soon become coherent. As we before said, the fluids which exude from the vessels in inflammation not only pass from the free surface, but infiltrate the membrane itself, and thus the tissue becomes softened. We find, on examining a case of acute peritonitis, how very readily the intestines are torn ; and if we take a portion, and gently incise the peritoneal coat all around, we shall easily be able to separate it from the muscular layer beneath ; it is important to remember this softness of an inflamed organ—it indicates the thorough change in its nutrition. The exudation may vary in kind : in acute sthenic peritonitis it is *solid lymph*, and is found covering the intestines and the abdominal parietes, and forming a layer over the liver and other organs. In other cases the peritonitis is asthenic, and the exudation is *purulent*, and then none of the signs we have mentioned are so well marked, but a quantity of milk-white fluid is found in the abdomen, particularly in the depending parts within the pelvis. Between these two extremes of solid and fluid there are many intermediate conditions ; thus two coils of intestine may appear to be adherent by lymph, but when separated some fluid may be found to have collected between them. If the whole abdomen is affected, the case is generally quickly fatal ; but we have seen one remarkable case where life was prolonged until the spaces between the coils of intestine were converted into abscesses, which at numerous points had burst into the canal. When the inflammation is local, it may be recovered from, or cause death more slowly.

In a case of fatal acute peritonitis we should always at once proceed to look for the cause of it ; for it will be found, in most instances, to be due to a previous disease of some organ which the membrane covers. We know the peritoneum either wholly or partially envelops the various viscera in the abdomen, and therefore if active disease be progressing in these, the peritoneum is apt to become involved, so that an inflammation of it ensues. The disease of any organ may be of a slow character, so that when the peritoneum is reached it is only locally affected, and, a small amount of lymph being effused, the resulting inflammation is confined to that spot. We thus find the liver adherent to the diaphragm, while an abscess is circumscribed between them ; occasionally, also, we find an abscess on the left side, which has resulted from disease of some organ in that part, as, perhaps, a perforation of the stomah ; or we find coils of intestines containing an abscess amongst them, or in the pelvis a local inflammation with similar results. If, however, an organ should suddenly rupture, and some of its natural secretions or other irritant fluid be poured out, universal and fatal peritonitis is generally set up. Thus abscess or hydatid in the liver, perforating ulcer of the stomach, perforating ulcer of the intestine (which is generally of the typhoid kind), ulcerations from old-standing organic disease of the intestine, escape of intestinal calculi from the cæcal appendix, or of any extraneous body which may have lodged there—all these are common causes. As less frequent causes we may mention escape of gall-stones through the ducts, rupture of ovaries, and various diseases of the genital organs ; and cases are related of its occurrence from worms perforating the bowels. Owing to various local circumstances suppuration may continue a long time behind the peritoneum without inflammation following, though a peritonitis may at last ensue. Suppuration of the kidneys very rarely penetrates to the serous membrane, and pelvic cellulitis may long continue without the peritoneum becoming involved. The extent of inflammation depends often upon the nature of the irritant which first sets it up; thus if urine is extravasated into the cellular tissue, death often speedily follows: the peritoneum may then become involved without any urine having actually escaped into its cavity, as is sometimes seen after the operation of lithotomy, where an inflammation around the bladder from extravasation of urine has lit up the same disease in the peritoneum. The same results follow if an organ be ruptured by violence; and this especially when the secretion or contents of the organ are extravasated, so that bile, urine, or food, or fæcal matter escapes from its place; but it is remarkable that blood has little tendency to cause peritonitis; a large effusion of this fluid may be fatal at once; but if the organ from which it proceeds be not mortally injured, the blood does not cause severe inflammation, but undergoes changes in the peritoneum, associated with a slight effusion of plastic lymph. This comparative innocence is, no doubt, due to its having a much closer affinity to the natural secretion of the membrane than any of the other foreign products. If no local cause be found for the peritonitis it is generally the sequel of Bright's disease.

*Chronic peritonitis.*—We have already said that acute inflammation of this membrane is very rarely idiopathic, but is dependent upon some anterior disease or injury of one of the organs which it covers. Chronic peritonitis, on the other hand, generally results from some morbid or altered condition of the membrane itself; although even here there is frequently some other element present to determine the inflammatory process, as tubercle or cancer. Occasionally, however, cases are met with where none of these adventitious matters can be detected, and then we are obliged to call the case one of simple chronic peritonitis. Sometimes the whole of the organs may be matted together, and thus, when the anterior abdominal walls are carefully removed, the bowels will be found united into a mass; the colon also adherent to the stomach, this to the liver, and the latter to the diaphragm; and at the same time the pelvic organs in firm union. When a slighter process of this kind has been present, there may be no exudation from the surface, but only into the substance of the membrane itself; and thus, although no adhesions may be found, the peritoneum is everywhere thickened and of an opaque white colour, the omentum also thickened, and the mesentery shortened and indurated; the surface of the liver, spleen, &c., being at the same time covered by a dense white tissue. In all such cases, however, there is probably some constitutional vice antecedent to the peritonitis.

There is a rare form of affection seen sometimes in children—an *ascites*, arising from a chronic peritoneal affection; but we have never met with a fatal case to examine; the disease being probably strictly analogous to a chronic pleuritic effusion, and of the nature of a catarrh. It has been described by some authors as occasionally occurring in the adult.

*Local peritonitis.*—Although the peritoneum is very ready to inflame all over when injured, yet sometimes inflammation is limited to a greater or smaller part of the sac, either through the slightness of the irritation or through the gradual approach of a more severe irritation. A grave inflammation approaching is preceded by a wave of milder irritation at its outskirts which induces plastic products that close the peritoneum at the affected spot. Slighter irritations cause, on a surface, a plastic lymph which requires only a few days to convert it into firm and organised connective tissue. The peritoneum over irritated parts partakes of the irritation; for instance, the peritoneum over syphilitic formations in the liver, or over a liver full of miliary tubercles, or over the whole liver in persons who drink excessively, may cover itself with a plastic coat, which, if the irritation is feeble, may not adhere to parts around, as it does if more severe. It may spread down to the stomach, omentum, colon, &c. A further excitation then supervening, as through tapping, &c., may set up acute and general peritonitis.

Again, another region obnoxious to the like local inflammation is the female pelvis, where, unfortunately as it proves, the genital apparatus lies exposed in the peritoneal cavity. Through syphilitic or other irritations the internal genitalia inflame, and then we have a local peritonitis, just as about the liver, but with a greater tendency to suppurate and spread generally over the whole peritoneum. If the inflammation is slight, the end is an adhesion of the parts, such as we often see in prostitutes; if greater, the inflammation may be general and fatal. Another obnoxious part is the neighbourhood of the stomach, through its liability to ulcers, which approach the surface and produce either a simple plastic peritonitis with adhesions to adjoining organs, or else, through the irritation being greater, an abscess; this bursting may produce a general peritonitis. Again, the lower end of the ileum is another of these dangerous parts; for it is here that typhoid, or, less frequently, tubercular ulceration within the bowel, brings risk of accidents just similar to those caused by ulcer of the stomach. The cæcum and its appendix, too, have a notable proneness to set up inflammation around them, and so the rectum in a less degree. These are the chief starting-points of local peritonitis, but it may arise from inflammations of the bladder, spleen, kindey, &c., or of lymphatic glands within the abdomen, or by abscess in the abdominal wall, &c. Hernias and their treatment we of course, cannot forget. If we examine the body some time after the operation for hernia we generally find a knuckle of bowel or portion of omentum, more or less adherent, near the opening of the sac. We have twice met with a curious mass composed of the lower end of the ileum, several coils of which were adherent by ancient well-organised areolar tissue, a ball being thus formed of the size of a cocoa-nut. In one of these cases there was an old history of hernia. In the other there had been no hernia; the position suggests that the fœtal vitelline duct was concerned in the matter. In neither case had it produced any inconvenience. Over cancers we often find a soft layer of recent plastic lymph; yet it is more rare to find old adhesions. It seems as if such peritonitis were often a part of the final break up. Indeed, we sometimes observe that a degree of peritonitis seems to start up in the dependent part of the peritoneum in cases of extreme exhaustion from very chronic disease. Thus, we have found several times, under these circumstances, a little pus in the lowest part of the pelvic peritoneum, or in its subserous tissue, or in the bottom of a harmless hernial sac. It appears as though the same thing occurs in the peritoneum as in the bronchial tubes, &c., in long-lasting moribund states, that is, the free secretion corrupts and becomes irritant to the surface in contact. Such a cadaveric peritonitis of dependent parts we have seen not infrequently.

We have seen inflammatory effusion limited to the lesser bag of the peritoneum so as to form a large cyst in a case of injury to the pancreas, and in a case of old hydatid abscess.

The old adhesions that result from local peritonitis are as common as pleuritic adhesion, but they are apt to be attended with more serious consequences. In many parts they are of no importance, as, for instance, when they connect the liver with the diaphragm; but by constricting the intestines such bands may give rise to fatal results, as may be seen in some of our specimens; a band of adhesion passes down over a portion of bowel and directly constricts it; or joined by its end to some other part, it may produce a loop through which a coil of bowel may slip. Again, it not uncommonly happens that the cæcal appendix or a cæcal diverticulum of the ileum becomes adherent at its end and forms a loop through which intestine may pass. Again, other organs, besides the intestine, may suffer, as when the chronic local inflammation causes adhesions of Fallopian tubes, ovaries, &c. In several cases of extra-uterine fœtation which we have seen the omentum has been adherent to the broad ligament, as though its union had disturbed the action of the Fallopian tube. Still it is especially as to the intestine that these adhesions are important.

We may often find in fatal jaundice a union of the several organs about the region of the gall-bladder, as the colon, duodenum, and gall-bladder with its ducts, all firmly united by dense fibrous tissue; on cutting them apart, a chronic local inflammation is seen to have occurred, and the cause of all is probably a gall-stone which may be found in the bladder or ducts.

*Hæmorrhagic peritonitis.*—We have already spoken of blood being found mixed with the inflammatory exudations in pericarditis and pleuritis, and now we must notice that the same sometimes occurs in the peritoneum. On first opening the abdomen we are surprised to see effused blood covering the intestines and other parts; but on closer examination we discover that it is merely lymph coloured by blood, having the same origin as the hæmorrhagic pleurisy before mentioned.

*Tubercular peritonitis.*—This is not so frequent as appears to be supposed. It often excites surprise when examining cases of pulmonary phthisis to see tubercular ulceration in the bowels so extensive as it often is, and yet not once in a hundred times lighting up tubercular peritonitis, though, as we shall see, it nearly always affects the subperitoneal tissue. Tubercular peritonitis occurs in a rather different class of cases, and it is generally a primary affection. It is apt to ally itself with tubercular pleurisy and pericarditis, and we may find all the serous membranes thus affected when the parenchymatous organs escape. Generally, however, there is tubercle elsewhere, although the tubercle of the peritoneum is the main disease. It appears under at least three different forms. The first may be called tubercular ascites; the surface is free from adhesions in the whole or nearly the whole of its extent. The tubercles form small knots of sizes from that of dust to swan-shot; they are thickest on the under surface of the diaphragm, the intestine suffering less. In these cases there is generally a great quantity of more or less turbid liquid effusion in the cavity, and clinically they are of the nature of inflammatory peritoneal dropsy. This form of disease is very like cancerous peritonitis, which we shall presently describe. Sometimes we can scarcely tell whether the little knots are tubercular or cancerous in their nature. At other times they are quite certainly tuberculous; in no other place do we see tubercle and cancer so closely approach each other in likeness, except, perhaps, in the lymphatic glands. The tubercles on the peritoneum may often be seen to be in close connection with the blood-vessels, and composed of a mass of cells.

The second form, also rather rare, is latent peritoneal tubercle; such cases are clinically very obscure. The patient wastes away, and has some indefinite local abdominal symptoms, say vesical irritation or irregular annoyance of the alimentary canal; treatment may be directed to the bladder or to symptoms resembling ileus, but after death one finds the peritoneum studded with tubercles in the midst of a soft semi-organised pellucid matter by which its surfaces adhere. The local irritation is then found to be due to the casual collection of a mass of lymph about the bladder or rectum or other suffering part. In one case the stomach was covered on its anterior surface with a thick layer of tuberculous matter.

But the third form is the most frequent, and that which is best understood as tubercular peritonitis. In fatal cases of this kind we find the intestines inextricably united together, so that any attempt to separate them will only end in their laceration and rupture; the abdominal walls are adherent both to the intestine and to the liver, rom which often they have to be cut, as well as from all the remaining viscera. The intestines become united, and the disease still continuing, ulcers form where the tubercular matter accumulates between the coils and the intestines are perforated, so that in the firmly united mass of intestine, many coils are found directly communicating with one another by perforations. Occasionally the coils are adherent around considerable spaces full of tuberculous pus which forms between them; into these spaces the contents of the bowels pass, and a local fæcal abscess is produced. When in this manner such perforations open into a free space, they present a peculiar appearance, owing to the contraction of the muscular coat, and the mucous membrane becoming everted, so that instead of exhibiting a small round opening, we perceive a raised swollen nodule like a rosebud. More rarely such fæcal abscesses attempt to make their way through the abdominal parietes, and perhaps burst or are opened, producing a fæcal fistula.

*Cancerous peritonitis.*—There is another form of peritonitis, where the morbific cause is cancer. The characters of the disease closely resemble the first form of tubercular peritonitis, the liquid products being more profuse, while the lymph on the surface is less in amount, the intestines being free, so that the case is often clinically a simple ascites; many of them are mistaken for hepatic dropsy. The disease is remarkable, also, in its being generally local; other parts of the body may not be affected with cancer, nor even, often, the abdominal organs themselves. It is this which renders the disease so obscure during life. The sole symptom generally being ascites, it is only in the absence of other causes for the dropsy that a suspicion is created as to its nature; but if the fluid be drawn off by tapping it is generally found tinged with altered blood, so as to be brownish or purplish in colour. This, as in the liquid of a hydrothorax, is suggestive of cancer. It is a form of disease frequent enough, though not much described, but it was always pointed out by Dr. Addison in his lectures. On opening the abdomen, a quantity of slightly bloody serum is found, and the peritoneum is seen covered with a number of hard tubercles, the omentum infiltrated with the same products, and the mesentery hardened and contracted. The thickening of the latter parts is often very remarkable, so that they form solid masses an inch or two thick, the omentum is drawn up to the colon, forming a hard ridge beneath it, which should be felt for during life in doubtful cases. Hard nodules are sprinkled over the intestines and abdominal walls, chiefly the under surface of the diaphragm and the flanks; these nodules, on microscopic examination, are often found to consist of fibres with a few spindle-cells, so that the disease might be called fibroma, and might be thought of in connection with keloid rather than cancer; the disease is called peritonitis because of the thickening and contraction of the serous membrane showing that a chronic inflammation has been going on; the same association of new growth with inflammation which we see in tubercular peritonitis, phthisis, &c.; likewise, as in such cases, generally, it is still a question how far the new formation is necessarily antecedent to the inflammatory changes, so in the present instance the same difficulty occurs. There can be no doubt, however, that in all such instances the process is one, in great measure, of chronic inflammation, but peculiar, owing to unknown circumstances; and, in the present case, putting on a cancerous form, as it does tuberculous in others. A quasi-inflammatory origin of this kind of cancer is often strongly suggested through its arising secondarily from cancers of the sigmoid colon, or ovary, stomach, &c., as though the cancerous action had spread from these parts swiftly over the whole peritoneum in a way that can only be compared to inflammatory activity; we have sometimes found cancerous knots in bands of old false membrane in the peritoneum. If fluid be present in the peritoneum, we can more readily understand how any new products can be diffused through the whole cavity. On the other hand, cancerous peritonitis may dip into and invade the intestine. This form of cancer is much more common in women than men, and occurs soon after middle age. Its limitation is very remarkable. In one of the last cases which occurred here, all the organs were matted together, and masses of growth existed in omentum and elsewhere. The liver, though covered with nodules, was unaffected, and the mass in the pelvis contained the uterus, bladder and rectum quite unaffected. On the other hand, a case which at first appeared to be diffused cancer of peritoneum was found associated with a cancerous tumour in the bladder.

**Morbid Growths.**—*Cancer* frequently involves the peritoneum when it affects any viscus beneath and reaches the peritoneal surface. The cancer germs may then be cast into the peritoneal cavity, and without exciting a general cancerous peritonitis may settle down to the bottom of the cavity about the pelvis, there setting up cancer in the form of little knots, which are called "daughter tubercles" by Virchow in relation to the mother tubercles they came from.

*Tubercle* may be often found in the peritoneum in cases of general tuberculosis, especially in the omentum, when the miliary tubercles often accompany the vessels, but its especial importance arises when it is primary and accompanied by an inflammatory process, as already described.

*Colloid.*—This kind of cancer is most frequently met with in the abdomen. Its general seat is in the neighbourhood of the stomach, colon, and omentum; or it is connected with a similar disease of the ovary. In the slighter cases it occurs merely within the walls of the stomach, &c., as will be mentioned under the several organs, but it shows itself apt to spread in the serous membrane. Thus the peritoneum may sometimes be found covered with these gelatinous masses growing from its surface, and the omentum immensely thickened, and converted into a long, flattened, hard tumour. A section of this would show the interior to be white, and having somewhat of a honeycomb appearance, consisting of a fibrous network containing the colloid matter within it. Sometimes this has formed in such large quantities that when the abdomen is opened several pints of it have poured out. In these cases its seat appears to be the subserous coat, involving also the peritoneum, and the walls of the hollow organs, as the stomach. In a beautiful and rare specimen in our Museum the liver is implicated in the disease. When examined, we find it composed of a network of delicate fibres, somewhat like the tissue of the lung, only finer and softer, and the spaces larger. In these spaces, or loculi, the white transparent gum-like colloid matter is contained. The microscope shows cells or nuclei in its midst which much resemble mucous corpuscles, but where the structure is firmer the spaces may be found filled with clusters of cells having all the ordinary epithelioid characters of cancer cells; all gradations from these cells into the mucous matter by "mucoid degeneration" may be met with.

Sarcomatous tumours are sometimes met with in the abdomen, and more usually in the pelvis, also occasionally lipomatous tumours. These may be so soft and fluctuating as to be mistaken for fluid. Sometimes where an ovarian cyst has ruptured into the abdomen, the peritoneum is seen to be covered with papillary growths like those in the cyst. The question has arisen, therefore, whether germs had not escaped from the ovarian cyst and found a soil for their growth in the peritoneum, in the same way as apparently an epithelial growth in the throat may let fall some of its germs into the lungs and there grow.

**Morbid Contents.**—*Blood.*—This is constantly found in the abdomen as a result of rupture of an aneurysm, or of injury to the liver, spleen, &c.; we have seen seven ounces result from bleeding of a hepatic cancer. The blood may be mixed, as we have just said, with inflammatory lymph. It is often a question of great importance to ascertain the extent of hæmorrhage which will prove fatal. The bleeding into the abdomen in cases of aneurysm affords an opportunity of measuring the fatal loss. We have found generally four to five pints, once only three. But in a case of aneurysm bursting into the thorax we once found seven pints of blood; there was, however, here reason to think that the hæmorrhage had occurred on two or three occasions, when a much larger amount of bleeding can be borne than at once. Where the amount of blood effused has not been sufficient to cause death, little or no peritonitis follows, but various changes take place in the blood; the fluid matter is absorbed, the fibrinous part adheres to the serous membrane, and the colouring matter undergoes changes to a darker colour. In the brain, it may be remembered, it becomes lighter. On the intestine, however, it becomes very dark, and in time almost black. In rupture of the liver or spleen, if the veins be opened, the blood is portal, and of a pitchy character.

A *black pigment* is often met with in the peritoneum, especially in that lining the pelvis; there is then generally some evidence of a former peritonitis; and as in inflammation some blood is often found effused with the lymph, it is probable that in these cases the source of the colouring matter is hæmatine. Occasionally the intestines are found uniformly black, at other times spotted. Such a condition sometimes occurs in connection with the deposition of pigment in other parts.

*Air.*—We have already alluded to the subject of air in the serous cavities as the result of secretion or decomposition. We have never seen it in the pleura, unless with clear evidence of its entry from without, and never an undoubted instance of it in the pericardium. In the peritoneum it is frequently found after perforation of the intestine, when the gas has clearly escaped into the cavity. In one case, which occurred here, it was evident that gas had existed in the abdomen for two or three weeks before death. The abdomen fluctuated, showing fluid, but at the same time was boggy and tympanitic, and on shaking the patient a splash was heard. On post-mortem examination a similar sound was heard on succussion; and on cutting through the walls a gush of air came out, the intestines were found contracted at the posterior part, and covered with lymph in which blood was mingled, as just now described. The source of the air was not satisfactorily made out, whether due to a decomposition of the blood and other products of effusion, or whether there had been an escape from the intestine. It is very likely that the latter was the cause, though, from the subsequent matting together of the intestine, it was impossible to make it out.

*Chyle* is sometimes found in the abdominal cavity from obstruction of the thoracic or lacteal ducts. In vol. vi of the 'Guy's Hospital Reports' an account may be found of a young man who had eight quarts of milky fluid in his abdomen, which proved to be chyle. After death a large tumour was found pressing on the mesenteric glands. Also in vol. xix of the 'Path. Trans.' Dr. Ormerod relates a case where fourteen pints of milky fluid had been removed from the abdomen of a man, the subject of a large tumour.

*Loose bodies.*—These may be of various kinds, as *biliary* or *intestinal calculi*, which may have escaped from their respective canals; it is said also that a loose *uterine tumour* has been found, and this we can well believe from a case we once saw where a large tumour hung by the thinnest possible pedicle. In most instances, however, the loose bodies are small and of a definite character, and have, no doubt, one source. They are oval or round and flattened, resembling a bean, enclosed in a capsule, and within composed of fibrous tissue and fat. Several specimens are preserved in our bottles, some loose and others attached. The latter are seen in course of development in the appendices epiploicæ; these latter processes become altered in structure and then drop off, constituting the loose tumours. One such body is described by a surgeon as being found in a hernial sac when he proceeded to operate, and one still attached caused strangulation.

*Hydatids* are sometimes found loose in the abdomen, or are developed between adherent organs; one, between stomach, colon, and liver, was remarkable, as the man fell twenty-five feet and broke his pelvis, yet did not burst the large hydatid.

## STOMACH

**Malformation.**—The stomach may be absent in unviable abortions, or it may be congenitally small. Some cases of hour-glass contraction —presently to be mentioned—are probably congenital. The stomach may be congenitally misplaced in the thorax through an opening in the diaphragm.

**Dilatation.**—This is generally a secondary affection, and is seen especially in cases of obstruction of the pyloric end. Just as the stomach contracts when little food enters from disease of the cardiac end, so it dilates in cases of scirrhous pylorus. In some cases of this affection which we have examined, on opening the abdomen the stomach was the only organ seen, being immensely distended and reaching to the pevis; and we may sometimes observe in the living subject the outline of the stomach thus distended from the great accumulation of fluid within it. Sometimes the distension is of a paralytic nature; and such paralysis we have twice known prove fatal after surgical operations. Sometimes the cause of the paralysis could not be found, but in one case where the dorsal vertebræ from the fourth to the tenth were involved in a carious abscess which implicated the splanchnic nerves, the distension was immense; we shall presently speak of general distension of the intestines from diseases implicating the solar plexus.

*Acute dilatation of the stomach.*—Dr. Fagge* has shown that enlargement of the stomach may occur as a primary and fatal affection. He describes cases where patients were seized with vomiting, bringing up a large quantity of fluid, accompanied by extreme tympanites, with collapse and death in a few hours. The vomiting was of the same kind as regards the easy throwing up of a large amount of secretion as is seen in cholera. Dr. Fagge regarded this affection as one of acute paralytic distension. Dr. Goodhart† further investigated the subject, and found that occasionally we meet in the post-mortem room with a very great enlargement of the stomach, which he thinks may be one of the causes tending to a fatal end. Bearing upon this Mr. Henry Morris related to the Pathological Society the case of a man who was under his care for disease of the ankle, and who, after an operation, was seized with vomiting, in which he brought up several pints of fluid and died in a few hours. The stomach was found greatly distended but otherwise healthy. He called the affection *acute gastrorrhœa.* It is not at all improbable that some of the cases related by the older authors, as gout in the stomach, were instances of acute distension.

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* 'Guy's Hosp. Rep.,' series 3, vol. xviii.

† 'Path. Trans.,' vol. xxxiv.

**Contraction.**—When little food enters the stomach it shrinks of course towards its œsophageal attachment, hence when gastrotomy is performed for stricture of the œsophagus the stomach is generally opened much nearer the pylorus than is expected. A similar contraction too may sometimes be seen where cancer is diffused through the coats, or when there is extensive ulceration and thickening of the walls. We also meet with stomachs in the state called hour-glass contraction. They appear as if a ligature had been passed around their middle, dividing them into two portions; and sometimes the peritoneal coat is thickened at the same spot, suggesting the site of a healed chronic ulcer.

Dr. Carrington* has collected several cases of this kind. In some there was a cicatrix as of an old ulcer, in others there was no trace of disease, and he therefore called them cases of congenital constriction. He believed there might have been ulceration in infancy of which no traces were left, but he suggested that the hour-glass form was a reversion to a type of stomach of some of the lower animals. Mr. Bland Sutton objects to this view, as he says there is no such approach to a division of this kind in the fœtus, and it is in no way comparable to the complex stomachs of ruminants. He maintains that we must always bear in mind that a reversion consists in the reappearance of a lost character or retention of a fœtal condition. More recently Mr. Roger Williams has investigated the subject by the light of other cases, and finding no disease in the mucous membrane, contends that the contraction is congenital. He maintains that both in the anatomy of the stomach and in its physiological working there is a tendency to a separate action in the two portions of the organ, and that as a result of this it may permanently put on the form of two distinct pouches.

**Injury.**—Wounds of the stomach leading to evacuation of its contents are generally fatal. Remarkable exceptional cases, however, occur, where the wound becomes adherent to the abdominal walls, and closes; and also where the edges unite with an external wound, and a permanent fistula remains, as in the well-known case of Alexis St. Martin, and a few other recorded examples. There was a man in the hospital some years ago, in whom there was little doubt that the stomach had been wounded by a stab with a knife, and yet he recovered; also a similar case which occurred at the same time elsewhere, in which it was almost certain that the stomach had been opened. We met also with another case where the stomach was torn through for an inch length over the spinal column in a man who was run over. He lived two weeks and then died of double pleurisy, but the rent in the stomach was adherent to the liver, and was undergoing cure. Sometimes we see a partial laceration of the coats of the stomach, as in one of our specimens, where the mucous membrane is detached and lacerated, a long rent being seen on its inner surface. It came from a boy who was run over when the stomach was full. In another case the peritoneum was torn and abraded in several places, but the mucous membrane was entire. We have also on our shelves a stomach with a laceration, which is supposed to have occurred during the act of vomiting; but this, we think, is very doubtful. The stomach may be displaced into the thorax through a ruptured diaphragm, producing *diaphragmatic hernia.* This can only occur on the left side, when the organ with some of the intestines may escape into the chest. In some instances, where there has been no history of accident, it has been thought that it might have arisen from disease; in one specimen the misplacement was congenital. In a specimen of stomach where gastrostomy was performed eight months before by Mr. Howse, the mucous membrane is perfectly healthy, merely showing a puckering around the opening.

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* 'Path. Trans.,' vol. xxxiii.

**Congestion.**—We do not know any more important morbid condition of the stomach than congestion, for it is only by a proper knowledge of this and similar states that we shall be able to recognise the results of poisoning by irritant substances; and even after long observation we often find it very difficult to pronounce in any case whether the organ be inflamed or not, seeing that the ordinary post-mortem appearances are so varied, according to the modes of death and conditions of the organs, especially the presence or absence of food. What makes the difficulty greater is that congestion and inflammation pass into one another, and that the latter is probably more often present than is usually believed. The example we particularly wish to speak of is the stomach in heart disease, which will be frequently seen in the post-mortem room. If the cardiac affection be of an obstructive kind, so that there is great congestion in all the organs of the body, the mucous membrane of the stomach is affected in common with other structures, and consequently presents a very remarkable appearance, and one which would be more likely to be regarded as acute inflammation than many cases of the action of irritant poisons. The interior is of a dark red or purple colour throughout, and on close examination this is found to be due to a hyperæmic state of the vessels, or true capillary injection; and, besides this, there is a large mucous secretion from the interior of the stomach, the whole being sometimes found covered with mucus of so tenacious a character that a strong stream of water is barely sufficient to wash it off; there is also sometimes an effusion in the mucous membrane itself, and in the submucous tissue, and then the whole wall appears thickened, and the organ feels fleshy and heavy instead of membranous; when we remember the epigastric pain, sickness, &c., sometimes attending heart disease, and then look upon such a condition as this which is found after death, we can hardly doubt that this extreme congestion has actually passed into an inflammation. Sometimes it presents a slate colour in patches or in an arborescent form. This arises from the pigment of broken-up corpuscles. The ulceration which is sometimes found in conjunction with it we will presently mention. Thus the stomach in heart disease approaches much more nearly to an inflammation than many cases where an irritant poison has been swallowed ; indeed we have never seen any case of the latter which at all equalled it, and therefore we should offer a warning not to pronounce too hastily that an irritant poison has been taken through any amount of redness or of mucous secretion that is found ; for we have never yet met with a case where any such condition could have warranted the suggestion of such a poison unless the fact had been previously known ; indeed we believe it is only when corrosion has been produced that we can speak with certainty of the action of poison from the anatomical state of the stomach ; a chemical study of the contents is always essential.

**Gastritis.**—*Acute catarrhal.*—It is remarkable how opinions have varied, and do still, with reference to the existence or frequency of gastritis. Formerly, when morbid anatomy was less cultivated, the various symptoms denoting severe stomach derangements were attributed to inflammation, and gastritis was one of the most frequently recognised disorders. Subsequently, however, when the viscera were more examined, and some tangible or very visible alterations were required to prove the existence of a morbid process, gastritis came into disrepute, and some eminent pathologists have denied altogether the existence of such a disease ; failing, more especially, to find alterations in the stomach of drunkards, and others, where inflammations had been thought so often to occur. It is probable, however, that this opinion was due to the absence of suppuration, and such well-marked results of inflammation, and the non-recognition of minute changes in the mucous membrane. The membrane, however, in such cases might show a milky opacity, at the same time being soft and thick, and more lacerable than natural, but its state microscopically would show very little that is characteristic. The epithelium of the follicles might be swollen and cloudy, and fall out coherent like renal tube casts. If the inflammation be severe, lymphoid cells of an inflammatory nature may be found between and under or even inside the tubes. Although the structural evidence is weak, there can be little doubt that acute gastritis is a frequent affection. Thus the stomach in cardiac disease already mentioned shows a *subacute inflammation*, as evidenced by the hyperæmic condition of the blood-vessels and the mucous secretion, consisting of leucocytes and blood-corpuscles ; and such a state, though less marked, may be often met with in other forms of disease, as in morbus Brightii, &c. ; and in cases of Addison's disease, where great irritability of stomach has existed, some evidence of gastritis has also been present. If we notice the symptoms attending such cases as these, we cannot do otherwise than suppose that an inflammation occurs. The frequency of stomach derangement in common catarrh, where the bronchi are thought to be the parts only involved, would suggest it as a not uncommon complaint; we have often found in cases of acute pneumonia and other febrile diseases the stomach in a state of minute reddening, its surface coated with tenacious and sometimes semi-opaque mucus, different in character from the natural clear mucus of the stomach walls, and in great excess of the normal quantity; this corresponds with the loss of appetite which is experienced in such febrile states. When the microscope cannot be used it is rather by these products of the inflammation than by any redness that we should be guided in judging of a catarrhal inflammation, for inflammatory redness of the stomach or of the skin may quite disappear after death. We should always examine the stomach, and become well acquainted with its morbid appearances.

*Phlegmonous gastritis*, or suppurative inflammation of the stomach wall, is rare; we have met with two instances in the form of submucous abscesses, some of them opening into the cavity and associated with suppuration in the track of the portal vein. In one of these the rectum showed similar abscesses. In another case the suppurative inflammation was diffused, with thickening of the coats. Although this is a rare disease, yet thirty cases were collected by Ackermann, who found that puerperal pyæmia appeared to be its more frequent cause; the state in one case was compared by Virchow to carbuncle. It is apt to set up pyæmic abscess of the liver, which is a rare result of chronic gastric ulcer. In *anthrax* or *malignant pustule* the stomach as well as the intestines may show hard nodules and sloughy patches on its mucous surface.

*Croupous gastritis.*—This is perhaps still more rare; it has been seen by Sir W. Jenner in diphtheria. Dr. Delafield says "it is almost always secondary to typhus, pyæmia, puerperal fever, cholera, dysentery, the exanthemata, and irritating poisons. The false membrane is in small patches, and may line a large part of the stomach." He gives a case of idiopathic croupous gastritis, in a man of forty-six, fatal in eight days. Dr. Wilson Fox has seen it in phthisis. We have seen it in a case of gouty kidney, the false membrane lining the whole organ; also in a case of abscess of the liver, along with similar croupous colitis. Otherwise, with the exception of a few unimportant patches, we have no experience of it.

*Gastritis from irritant poison.*—We know nothing less than an actual breach of surface, or some chemical change in the tissue, which can decide us in declaring that a stomach has been affected by an irritant, especially since the hyperæmic state found in heart disease is believed, only with some hesitation, to have passed beyond the stage of congestion. In the case of strong acids, the so-called corrosives, a chemical change occurs in the mucous membrane, and a dark brown, greenish, or black mass results; we see how in many of these examples the poison was never carried round the stomach, or else it had become so diluted that its effects were lost; so that we observe the effect only at the spot where the poison first struck the inside on entering, this being about the middle of the greater curvature, opposite the œsophagus, and thus often only the pyloric half of the organ is affected. In a case of fatal nitric acid poisoning the corrosive action had leaped from the cardiac half to the pyloric end without much hurting the intermediate part. In the stomach of a child, who died from swallowing some soap-lees, the œsophagus is of a brown colour throughout, with one or two brown spots on the membrane; in cases of poisoning by sulphuric acid, the parts with which it comes in contact are blackened, and hardened into a thick mass; and in some cases the acid perforates the stomach. In one case two thirds of the stomach wall were completely gone, and the remainder showed no charring, or mere traces of it. The acid after escaping may involve the neighbouring organs, which become charred. In one case, where the woman lived several days after drinking diluted sulphuric acid, the whole mucous membrane was found lying loose within the stomach, and of a bright yellow colour. In a case of poisoning by nitric acid the interior of the stomach was of a curiously bright green mingled with dark brown and black. One affected by hydrochloric acid is coal black. But, though these varieties prevail, the effects of the several acids depend too much on their concentration to be quite characteristic. Caustic alkalies convert the mucous membrane to a tawny pulp; they often perforate the stomach. Carbolic acid produces a dry tanned-looking surface. In cases of poisoning by arsenic, the effects are not due to corrosion but to irritation, and inflammation may be slight; thus in one of the last cases occurring here, where death took place after ten hours, the inflammation was only characterised by the red colour and injection of blood-vessels, there being no ecchymosis, extravasation, nor any ulceration. In one specimen we see a patch coated with mucus containing the poison, where a circumscribed intense inflammation had occurred. In another case, that of a young man who survived six days, numerous ulcers were found. Various other conditions may be seen in our preparations and drawings. In cases of arsenical poisoning we have noticed an absence of the usual signs of decomposition, such as blackening and distension of the bowels, these being small, and of a pinkish colour with little gas in them; indeed very like the intestines in cholera. Arsenic directly prevents decomposition. Cholera sweeps away all the food that would decompose. The effects of a chronic inflammation and ulceration, leading to perforation, are seen in a specimen of a stomach affected by chloride of zinc. The woman, who was a patient of ours, drank some Burnett's solution, and survived twelve weeks. The organ is exceedingly contracted and ulcerated, and communicates by an opening with an abscess at the cardiac end. In poisoning by vegetable irritants, the morbid appearances of the mucous membrane of the stomach are very slight and fallacious, for it must be remembered that so many of these substances act on the nervous system to produce asphyxia, or difficulty of respiration, which tends to congestion of all parts, including the stomach. Thus in a case we lately witnessed, where the mucous membrane was of an intense red colour, all the organs participated in the congestion owing to the mode of death. In a case of poisoning by bitter almonds, which occurred here a short time ago, the mucous membrane was of a bright pink colour from vascular injection, which was regarded as the first stage of an inflammatory condition ; even this, however, might have been overlooked, had not the history been known.

Oxalic acid poisoning is speedily fatal, but in a case which extended over two weeks a gastritis was set up. The patient vomited a greenish-brown fluid, and after death the mucous membrane was found greenish yellow, injected and abraded in parts ; the pyloric end of the stomach thickened from infiltration of submucous and muscular coats.

*Chronic inflammatory, and other changes in the stomach.*—We just now said that the first study of morbid anatomy tended rather to the denial of such a disease as gastritis, but we have seen that in all probability it is not uncommon. In searching for the disease we labour under a disadvantage in that it is of itself not fatal, so that it is only found as a complication of more severe disorders such as phthisis, albuminuria, &c., whose symptoms often overwhelm the signs of the gastritis, and thus the anatomical changes which we see in the walls of the stomach often cannot be distinctly linked with clinical signs of gastritis. In chronic gastritis we find the mucous membrane more opaque and thicker than natural, its colour perhaps altered to a grey or brownish tint or even a black colour, through pigmentary change in extravasated blood. The mucous membrane may be rather tougher than usual, so as to allow of its being more easily detached by pulling, but if the inflammation has been more severe the submucous tissue is thickened, white, and fibrous, so that separation is more difficult. The surface is often in that condition called *mammillated.* This we constantly see in the post-mortem room, and we have a specimen of it in a bottle ; instead of the mucous membrane being smooth it is covered with a number of minute projections lying close together, as if the contraction of the submucous tissue had drawn the mucous membrane into this form. This is probably the cause, for we think it is not yet satisfactorily proved that it is due to adventitious structure, or is really morbid—at least not more so than the analogous case of *cutis anserina.* If we examine sections of an inflamed stomach hardened in chromic acid, we shall have no difficulty in seeing that important changes have taken place, although these do not usually amount to a disintegration of the structure. Thus we find the interstitial tissue between the tubes thickened in places ; if we examine patches of such thickening we find that the apparently new matter is made up largely of shrivelled tubes. At the same time the tubes become much less straight and parallel than they properly are, so that they look confused, and it is difficult to get sections of their whole length; their walls are thick, their contents are fatty and granular in many parts, and in some places quite disintegrated. Sometimes the mouths of the tubes are blocked up, and the deeper parts dilated into cysts. The condition of the solitary glands is not constant; sometimes they are large and ulcerated, sometimes they are wasted so as to be scarcely discoverable. We have not seen lymphoid tissue between the glands such as some describe in chronic gastritis. The whole of the changes correspond with the conditions observed in glandular organs, especially the kidney, in chronic inflammation, indeed chronic gastritis is often associated with granular kidney and similar chronic changes, just as acute gastritis is associated with acute pneumonia and other febrile disorders. Since Drs. Habershon and Handfield Jones first described this condition much attention has been given to it, especially by Dr. Wilson Fox, in very able researches; our own results correspond very nearly with those of these authors, but we have been much impeded by the unfortunate difficulty in obtaining good clinical evidence of the gastritis. Often we find much change where there were few symptoms, and little change where the symptoms led us to expect much.

Dr. Handfield Jones many years ago spoke of this change as "an infiltration of a low fibroid tissue loaded with nuclei or small cells amongst the tubes, which themselves undergo atrophy, so that at last the mucous membrane totally loses its tubular aspect and becomes a mere fibroid structure." In this degenerated tissue sometimes small cysts are found.

The mucous membrane of the stomach may be found affected by *lardaceous* disease; it then looks sodden and dull, and its surface is stained of a deep walnut colour by iodine. This condition is only found as a part of a very general lardaceous disease of the viscera, and as in other parts, the disease begins in the small arteries. This state produces a liability to catarrh, as in the kidney and elsewhere.

*Atrophy of the stomach.*—It has been stated by Dr. Fenwick and others that in idiopathic anæmia the mucous membrane of the stomach is atrophied. It has become thin, and when examined by the microscope the tubules are wasted and contain fatty granules.

**Ulceration.**—*Catarrhal.*—Ulceration, as an effect of inflammation, is not so common as might be thought, ulceration being generally cancerous, or else of that equally characteristic kind known as "chronic ulcer." It may occur, however, from the effects of poison, as we have seen, and sometimes may arise as scattered minute ulcerations in connection with acute catarrhal gastritis. Such a specimen we have where minute ulcerations are seen on the stomach.

We occasionally, but rarely, meet with simple *follicular ulcers;* they are small and unimportant.

*Hæmorrhagic erosion.*—This is a name given to an appearance of the stomach met with chiefly in those remarkably congested organs found in heart disease, of which we have already made mention; besides the extreme congestion, the mucous membrane is found destroyed in various spots, and occupying each of these is a layer of black coagulated blood. The blood escapes by reason of a superficial destruction of the tissue at the spot, and this destruction occurs, apparently, through degenerative changes in the parenchyma. It may be that the ulcerations happen in the dying hours, and thus the minute clots in the ulcers are recent. We recognise them in the deeply congested stomach, by seeing small dark spots or streaks, which, on closer examination, are found to be coagulated blood, occupying spots where the mucous membrane is deficient. Sometimes, however, this form of ulceration occurs long before death and causes hæmatemesis.

*Chronic ulcer* is a special form of disease, whose history has been, perhaps, more elaborately followed out than that of any other disease in the body. The mucous membrane of the stomach naturally endures the contact of the gastric juice with impunity; we shall presently mention the interesting inquiries into the causes that enable the stomach thus to resist self-digestion. It has long been thought that gastric ulcer may arise through a part of the stomach losing its resisting power, and being dissolved in the digestive fluid. Virchow gave this idea definite shape by supposing that the starting-point of the ulcer is an arrest of the circulation in the affected part. His comparison of the funnel-shaped figure of the gastric ulcer with the funnel-shaped area of distribution of a gastric artery is suggestive; and subsequent experiments, which show that ligature or embolism of the arteries of the stomach will produce ulceration, have proved that the gastric juice will dissolve not only dead pieces of the stomach wall, but also parts of it whose nutrition is much lowered. Again, the acid juice may be suspected of causing these ulcers, from the fact that they occur almost only in the stomach and in the portion of duodenum above the entrance of the alkaline bile. Yet we cannot accept this view as more than an interesting hypothesis, of partial application; for it fails entirely to explain the remarkable fact, that spontaneous ulcers are almost limited in the stomach to its lesser curvature. Again, the class of persons who are most subject to chronic ulcers of the stomach are not those in whom obstruction of the arteries occurs. We have now seen very many cases of wide-spread embolism of the different viscera in heart disease, and yet no ulceration of the stomach was present; while, on the other hand, subjects of ulcer have not generally any cause of embolism present. We have seen several cases of total obstruction of the portal vein without ulceration of the stomach. Further, the experiments of Dr. Pavy and others, which show that ligature of the gastric vessels will lead to digestive solution of the part affected, do not prove that the arrest of the blood is the cause of the solution; because such ligature must so damage the nerves, lymphatics, &c., of the part as to practically kill it, or nearly kill it, and thus reduce the result to the solution of a partially necrosed tissue, which is dissolved because its peculiar vital power is gone. Dr. Pavy's ingenious theory of the protection conferred by the alkaline blood, which protection he supposes to fail when the supply is stopped by ligature, is of too limited application to explain the resistance of gastric surfaces to self-digestion, for polyps and other low animals in whom there is no alkaline blood show an equal security from their own digestive fluids. Hence we believe that although it is proved that solutions of continuity in the gastric mucous membrane will arise through ligature of the arteries, yet this is not at all shown to be the cause of gastric ulcers. We are disposed to regard these as more probably due to nervous influence, and as having a close analogy with simple ulcer of the cornea.

These ulcers appear so constantly in the lesser curvature, which, it must be remembered, is a very small part of the whole stomach, that some peculiarity of this part must be sought out to explain the association. Sir W. Gull suggested that the fixity of the lower curvature during the movements of the stomach might create some irritation; but at present there is evidently much to be learned before we can explain satisfactorily this peculiarity of their position. They are mostly found at the posterior part of the lesser curvature and in the pyloric half, being very rare in the fundus. They are so often circular that the term *round ulcer* is given to them. They are usually solitary, but sometimes two or three may be met with together. They may be of all sizes up to that of the palm of the hand.

Sometimes the ulcers rapidly proceed to perforation. This is especially apt to be the case in young women, and particularly in young servant-maids. Such rapid perforation is almost always fatal, so that inspection reveals the ulcer in an early stage; its figure is then circular, or very nearly so; it looks as though a piece had been punched out, so like indeed that it is commonly called the *punched ulcer*. The opening is larger on the mucous surface, and gradually narrows toward the peritoneal surface; the edge is defined and even, and the outline abrupt and bold; usually no granulations or other signs of healing are present. This circumstance, together with the sort of people who suffer, suggests that the rapid course is due to some constitutional weakness. No bacilli have yet been found in them, as is sometimes the case in ulcers of the intestine. In a drawing an ulcer is depicted in which Dr. Habershon dissected out the branches of the pneumogastric nerve.

We occasionally meet with small gastric ulcers, perhaps entirely unsuspected in the fatal illness, the symptoms having been overwhelmed by those of the main disease. These we have seen as single, reniform, or circular erosions, generally near the pylorus; sometimes they are quite shallow, and a little blood extravasated in the mucous membrane around would give rise to the suspicion that hæmorrhage into the tissue, weakening it and leading to its solution, may be a cause of such ulcers. They are most often associated with Bright's disease.

When the ulcer is older and larger it has still a clean-cut edge in the wall of the stomach, but its base is in a more variable state, sometimes showing decaying tissue. This base or floor of the ulcer may either be the proper coat of the stomach, or else it may be the organ adjoining the stomach at the affected part, which organ has become fastened to the stomach in the spread of the inflammatory process, and has been invaded more or less deeply by the ulcer. Thus the pancreas usually (as these ulcers are mostly at the back of the lesser curvature), or the liver, or even the spleen, is invaded, or the adhesions are to the neighbouring intestines or the anterior abdominal wall, either of which may be invaded or perforated. If perforation take place slowly adhesions form, and the stomach may open into a circumscribed cavity, bordered by the diaphragm and spleen. It is noticed that when the ulcer is on the anterior aspect of the lesser curvature there is greater liability to a perforation into the peritoneal cavity; you then may find castor-oil and white mixture, &c., in the peritoneal contents; such perforation occurs in about 12 per cent. of all cases of gastric ulcer. A greater proportion of danger to life arises through hæmorrhage from invasion of arteries. Such hæmorrhage may be slight from smaller vessels, or of medium severity, gradually blanching the patient, or else it may be quickly fatal through the opening of a greater vessel, as the gastric, pancreatico-duodenal, splenic, or, in rare cases, the hepatic artery, or the portal vein.

This reminds us that *hæmatemesis* is spoken of as a distinct disease of the stomach, resulting usually from portal congestion, through heart disease, or cirrhosis of the liver. A specimen we have professes to be the stomach of a man who died of hæmatemesis, but in which no ulceration could be discovered; in other instances, however, the observer has been more fortunate, and one specimen shows how minute an ulceration is sufficient to lay open a large artery. Dr. Murchison has recorded two similar instances in which very small pore-like ulcers went directly into gastric arteries and caused fatal hæmatemesis; but sometimes the most careful search fails to discover, in cases of cirrhosis of the liver, the source of the bleeding, and we have to fall back on the theory of exudation, which, however, Cohnheim's experiments have now made more acceptable. In a case where a gastric vein was opened we found it varicose and the portal vein full of thrombus.

Several cases have been observed where the fatal hæmorrhage arose from the rupture of an aneurysmal dilatation of a blood-vessel within the ulcer, just as often happens in a cavity in the lungs.

Instances are occasionally met with of fatal hæmorrhage into the stomach and bowels of new-born infants arising from gastric ulcer. Dr. Goodhart has placed a specimen of this kind in our Museum.

Many stomach ulcers undoubtedly heal, as may be seen by examining the specimens in our Museum ; if small, leaving very slight trace ; but if larger, causing a puckered scar on the mucous surface, or even drawing in the whole of the coats so as to greatly alter the shape of the organ, causing hour-glass contraction. A very chronic ulcer may produce such contraction without healing, and so may not only deform the stomach, but cause perforation, as into the duodenum or other unusual direction. We do not think, however, that every puckered surface found at the lesser curvature implies a cured ulcer ; for it may represent the disease in process of formation. We have been too much in the habit of supposing that all great structural changes have resulted from an inflammatory and ulcerative process—that where there is a new fibroid tissue there must have been an inflammation ; or where a contraction, a previous ulcer. We have already shown a thickened syphilitic larynx with an irregular mucous surface, without ulceration ; and in the case of keloid in the skin the disease is always erroneously considered by the ignorant to be due to ulceration or burn ; and thus we think a puckered surface, or a withering of the mucous membrane, may be due to an exudation and contraction of the material in the submucous tissue, without any preceding ulceration.

A very remarkable case of recovery from perforation is published in the 'Guy's Hospital Reports' for 1846. A young girl became suddenly collapsed, but having revived had acute peritonitis and then recovered. She died some time afterwards, when old adhesions were found surrounding a perforating ulcer of the stomach.

We once found two bean-sized *syphilitic gummatous* nodes in the subperitoneal coat of the stomach.

*Tubercular disease.* — This is excessively rare, many pathologists having never met with it. We have, however, two if not three specimens of it in the Museum. One was from a child aged eight years, who died of phthisis and tubercular peritonitis. The stomach contained an irregular ulcer with flocculent edges near the pylorus, on the floor of which were distinct yellow tubercles, and also on the corresponding peritoneal surface. The other stomach was from a child, two years old, who died of general tuberculosis. The stomach had two ulcers on its posterior wall with raised edges, and corresponded in appearance exactly to the tuberculous ulcers in the intestine of the former subject.

**Cancerous Affections of the Stomach.**—We believe the different names which we give to many diseases imply merely that there are some distinctions between them, which make most of them easily recognisable ; but, at the same time, these so often approach one another in character, that no very definite boundaries can be made. Thus we meet with well-marked soft medullary cancer growing in the stomach and destroying it ; in another case a firmer growth, which may be called scirrhous ; in another a growth which appears simply fibrous. At the pylorus the last kind may be quite local, so that it is questionable whether the name cancerous can be applied or not. Combined with these forms we frequently have a gelatinous infiltration called colloid, which, however, may occur by itself; and there is also the disease called villous cancer.

There are all degrees of activity of growth and malignancy of gastric carcinoma; and these degrees are generally indicated by the hardness or softness of the cancer. The softer the growth, the more rapid and malignant. The harder and slower forms are more common at the pylorus, so that the term *scirrhous pylorus* is currently used for cancer of that part; although we do sometimes meet with soft cancer there. Sometimes the disease is of very slow growth, and on examination the appearance is of a thickening about the pyloric orifice, as though from some hypertrophy of the muscular coat and submucous tissue. One preparation came from a man who died of renal disease, but had some vomiting, and the pylorus was found thus thickened. We can scarcely indeed apply the term cancerous to it, nor even to one more marked example, where the disease existed four years, and caused the death of the patient; for we see it is quite local, and does not at all invade any of the surrounding tissues; moreover, the new material thrown out is very hard and fibrous, having no juice, as cancer should have. The section shows very well the appearance of a scirrhous pylorus; before it was opened, the enlargement of that part of the stomach produced a tumour of the size of a hen's egg, and it was moved by its weight considerably lower than its usual site in the abdomen.

In such cases it would be fair to question the cancerous nature of the growth, and even microscopic examination may show so large a proportion of fibrous tissue that a doubt may remain. Here perhaps more than at any other part of the body do we see gradations between simple fibrous tissue and cancer. There is no doubt that stenosis arises sometimes from the cicatrisation of a chronic ulcer, and this is the condition which has been cured by operation.

But a typical *hard carcinoma* of the stomach is less ambiguous in its characters. It appears as a solid hard thickening of the coat of the stomach, generally near the pylorus, tending to surround this, not passing into the duodenum, but extending towards the cardia, especially along the lesser curvature, and having generally a well-defined raised border. Section shows the thickness—about one half to three quarters of an inch in all—to be made up of the coats of the stomach in an altered state, but still recognisable. The muscular coat is best seen with its greyish pellucid bundles strongly marked, and separated by white septa; this forms nearly all the outer layer, and composes about one third of the whole thickness. The rest of the bulk, about two thirds of all the thickness of the mass, is composed of cancerous submucous tissue. The muscular coat may be quite transformed to cancer in the more central parts of the growth or the new substance may only separate the muscular bundles; in either way it reaches the subperitoneal tissue, puckering and nodulating the peritoneum; or causing a subinflammatory adhesion to adjoining organs, and finally an invasion of them. The glandular mucous membrane itself is often raised over the tumour, and generally soon ulcerated more or less; an excavation forming with a thick raised wall, and a more or less sloughing base.

In some of these forms great contraction of the stomach takes place. In a specimen described many years ago by Dr. Habershon the stomach is seen to be contracted to a very small size; whilst its walls are thick, and villous tumours are sprouting from the inside. In another case the stomach is about a third its natural size, and its walls are occupied by a sarcomatous growth except a thin band along the greater curvature. In another old specimen of Dr. Bright's the stomach is small, but the whole of the coats are thick, giving the organ the look and feel of an india-rubber bottle. The œsophagus is also hypertrophied.

The *medullary* form of carcinoma affects rather the cardiac orifice of the stomach (there is a notable tendency of cancer to affect the orifices; in this it differs from ulcer). The medullary kind of cancer has a more active transforming power, so that we may find the muscular coat altogether disappearing in parts, the cancer reaching the peritoneal tissue, and raising this in larger or smaller nodules. Medullary carcinoma, however, spreads with chief violence in the softer submucous layer, so that this is disproportionately enlarged, and may grow into great lobular masses, which are very liable to suffer from either deep ulceration or digestive action, so as to slough away in patches; giving then rise perhaps to extensive bleeding sores, or even opening large arteries, as the splenic; but except through such sloughing the bleeding in cancer is slight. This sloughing away of cancerous masses may perhaps reopen the food channel previously obstructed by their growth, and so bring great relief and comfort to the patient in this miserable disease. But the sloughing may extend through the whole wall of the organ, so producing fatal peritonitis. It is difficult to describe the loathsome appearance which the foul, ragged, sloughing masses in the interior of such a stomach sometimes display.

Gastric cancer, which is usually primary, originates in all probability in the gastric glands, and it is very apt to spread by contiguity. Thus, the liver may be invaded so that several square inches of its altered substance lie exposed in the stomach in the shape of a sloughing cancerous sore. The pancreas or pre-vertebral tissue, and even the vertebræ, may be attacked, or the colon, or the duodenum, or rarely other portions of the small intestine, may be opened. Such openings from the stomach into the intestine are more frequently caused by cancer than by simple ulcer. The lymphatic glands share in the cancer, and by them the biliary duct, the vena cava, or the vena portæ, may be compressed or invaded. We have seen the gastric and portal veins stuffed with such cancer. Besides this local extension to contiguous organs, secondary cancer is apt to appear in the form of round nodules in the liver or lungs, or on the mucous membrane of the small intestine, &c.; in fact, in the usual seats of secondary cancer, with especial preference for the liver.

The effect on the peritoneum is to cause a slight local peritonitis, or, in rare cases, a general peritonitis may be set up without perforation of the stomach by the cancer.

As to the organ itself, the remainder of its mucous surface is generally healthy. If the cancer obstruct the cardiac orifice the stomach is empty and small; if the pylorus, it is large, often very large indeed. If there is a considerable cancer of the pylorus its weight may drag that part down, so that it is found much below its natural position. This fact we must remember at the bedside, for the pylorus may thus descend almost to the pubes.

*Villous cancer.*—The cancer sometimes implicates especially, and takes its character from, the most superficial layer of the gastric mucous membrane, showing then a tendency to form processes which may grow and branch very freely, producing a richly clustered mass of dendritic vegetation, subject, however, to defacement by sloughing and ulceration. The structure of such processes, when minutely examined, is often very beautiful. Each is composed of a central blood-vessel clothed with well-formed "cancer-cells." The malignancy of some examples of this cancer was extreme, so that the growth invaded the liver by contiguity, a great mass of soft carcinoma spreading into it from the floor of the papillose ulcer. We shall presently speak of superficial villosities which have no such cancerous nature.

*Epithelioma.*—We have seen a few cancers of the stomach which showed, microscopically, the most perfect glandular structure, resembling, that is, the mucous glands of the intestinal wall. They begin in the epithelium, extend to the submucous tissue, and then invade the other structures. They are not common, but sometimes met with encircling the pyloric end. Such cancers look like medullary carcinoma; we shall describe them more particularly in their favourite seat in the large intestine. Secondary to such cancers we have seen similar glandular structure in the liver.

*Colloid.*—We have already mentioned this disease as especially affecting the peritoneum, and in the most marked cases we find the whole of that membrane, including the serous coat of the stomach, occupied by the deposit. Colloid cancer of the stomach is generally a compound disease; for example, a scirrhous cancer is associated with colloid, and when a section is made the new matter is found to be softer than ordinary scirrhus; on closer examination a gelatinous substance is seen mixed with it, and may be picked out with the point of a scalpel. In these cases the muscular tissue appears more destroyed than in carcinoma; the peritoneum is invaded too, and thus the disease is a more uniform infiltration of the stomach wall; also it spreads generally more widely. A section through the pylorus shows the general infiltration of a gelatiniform matter. A purer and simpler form of colloid is seen in other cases where no ordinary cancer appears in any part of the body; the whole of the coats are infiltrated with the jelly-like matter, the mucous membrane is raised, and on looking at its surface, it appears translucent from the pouring out of that substance beneath it. We have a remarkable example of colloid affecting the œsophageal end only. Colloid has little tendency to ulcerate, or to form secondary nodules in the liver, but is apt to light up general colloid disease of the peritoneum.

Dr. Pye-Smith has had lately a remarkable case of primary colloid of the stomach. A large growth projected into the cavity, and did not infiltrate the walls or the peritoneum, as is usual. The neighbouring glands were similarly affected, and the lungs were full of colloid material.

*Sarcoma.*—Occasionally, without any disposition to ulcerate, a growth may extend slowly within the gastric coats, and maintain its firm character, as though simply fibrous; but instead of consisting of simple fibre tissue, it is seen to be composed of nucleated fibres, like those which are known as spindle-shaped sarcoma. In our specimens we may see the organ immensely thickened throughout more than half its extent by this adventitious deposit. In one case the pyloric half is thus affected, and in another the half comprising the lesser curvature.

We have already spoken of fibrous cancer involving the walls of the stomach, but it is often difficult to say whether such cases are true cancer or simply sarcomatous. We use this term when the walls are slightly thickened, the muscular and submucous tissues being occupied by spindle-shaped cells converting the stomach into a tough hard vessel like an india-rubber bottle. In one of our specimens the walls are an inch thick. In another only half of the stomach is affected in this way. In an old preparation, where all the coats are much thickened and the œsophagus is in the same state, the term colloid is applied to the disease. In another the walls of the colon are thickened in the same way as those of the stomach.

In some instances there is black pigment in such sarcomatous growths forming *melano-sarcoma*, or, shortly, melanosis of the stomach. It may be secondary to melanosis of the eye or skin: it is very rare. It is sometimes seen dotting the mucous membrane.

*Myoma* is sometimes met with, usually as a hard, white prominent mass, into which the muscular coat expands; sometimes such a tumour projects considerably, forming a *polypus*, or several small polypoid growths. In one specimen we see one or two small growths consisting of fibro-cellular tissue, with an admixture of muscular fibre, and there were three such pedunculated tumours found accidentally in the stomach of an old woman. Their structure would entitle them to the name of myoma or *myofibroma*, and they correspond to the descriptions of this condition which are published. Cases are recorded where a polypus of this kind has been vomited up.

Virchow speaks of myomatous tumours of the stomach arising in the muscular coat, and growing either externally or internally. Dr. Kidd exhibited at the Pathological Society a case of fibro-myoma where one third of the surface of the stomach was involved. It consisted of a fibrillated tissue interlacing with long-shaped nuelei. Dr. Taylor has also lately placed in the Museum a large circumscribed growth of the same nature.

*Fibrous papilloma.*—We meet with specimens of villous growth of a very simple kind springing from the gastric surface. The microscope shows these villi to resemble very much the similar processes of the chorion of the ovum, and when the two are placed under water the resemblance is very exact. When of this simple kind they spring immediately from the mucous membrane; we shall have to show the same in the bladder; they have only the slightest fibrous basis. These growths are now described as *fibroma papillare*, and must be distinguished from villous cancer.

Small *lipomata* occasionally occur in the submucous tissue of the stomach.

*Adenoma* may occur in the form of local hypertrophy of the gastric glands or as a tumour. The tumours are small, white, and translucent. They do not give out a milky juice but a mucus full of cylindrical epithelium. They are composed of a tabular structure like normal glands. Dr. Cayley showed at the Pathological Society a large tumour of this kind growing from the inside of the stomach which he called lymphadenoma.

*Cysts and pouches.*—We spoke of a cyst in the œsophagus which we lately met with in the post-mortem room, and we have seen a very similar cyst of about the size of a walnut in the walls of the stomach. Also a pouch, which may have been originally a closed sac, but is now broken into; and in our Museum is a similar preparation dried. Cysts of small size from dilatation of the glands are not very unfrequent.

**Cadaveric Softening.**—As in describing many of the morbid appearances of the stomach we have referred to post-mortem changes, it will be convenient to speak next on this subject; and we must again speak of the importance of studying these changes, since they are much more striking than many appearances which may be due to disease. We know that the gastric juice after death acts on the coats of the stomach, to dissolve them in the same way as it does ordinary flesh; it has generally been thought that the juice retains its strength, while the stomach itself loses its vitality, and thus becomes subject to the solvent influence of its own secretion. Bernard, however, has shown (and Dr. Pavy has repeated the experiments) that the solvent power of the gastric juice is equally great on living tissues, hence it is thought that the reason why the stomach is not affected during life is due to the constant evolution of mucus and epithelium, which protect it; or, as Dr. Pavy believes, the alkaline blood in the coats prevents the acid juice from attacking them.

However this may be, the gastric juice after death will dissolve the stomach; but the juice appears to be secreted only by the stimulus of food, so that if the patient die with the stomach empty, it may be found several days after death unaltered, with its mucous membrane entire, and its rugæ prominent and unchanged.

If, however, there be food in the organ, and the digestive process going on, then the mucous membrane will be found to have undergone solution, and, in some very rare cases, all the coats will be found to be destroyed, leading to perforation. The extent of stomach affected corresponds to the quantity of food within it. The part affected depends upon the position of the body after death. As this is generally supine, it is the larger end of the stomach, at its posterior part, which is usually acted upon; and if the contents be removed, we can see by the change which has occurred to what height the food reached, the water-mark being accurately defined by a distinct margin; above this margin, in the pyloric end of stomach, which lies superior, the natural rugæ of the membrane are seen. In the most depending part which has held the food what is mostly seen is a softening and thinning of it, so that it looks like a layer of clear mucus, or there is a complete loss of the mucous membrane, when the submucous tissue is exposed; or this may be also removed and the muscular coat softened and thinned, or even the peritoneum perforated. The affected part differs from the rest of the organ, not only in thinness of its coats, but in being smooth from the loss of the rugæ; it also presents an appearance, from the action of the gastric juice on the blood and vessels, which is very characteristic and is resembled by no pathological state. We know that the gastric acid renders the blood black, and thus, when the vascular submucous tissue is exposed, large vessels are seen coursing along in it of a black colour; but the blood has exuded through their walls, and thus we see an irregular arborescent appearance of ill-defined black lines running over the dissolved part. This is seen in a preparation and a drawing; the latter showing admirably the process of digestive solution. In cases of perforation from digestion of all the coats, the contents of the stomach are found in the abdomen; we have already alluded to such a circumstance occurring in the œsophagus, with escape of the gastric contents into the chest; and occasionally the duodenum may equally suffer. Such a perforation is easily distinguished from a perforation through disease both by its position and character; its position at the cardiac end is unusual for ulcer; and as an ulcer arises in an inflammatory process, its edges will be found thickened from lymph, fibroid tissue, &c.; whereas in case of softening the opening is large, the edges thin, dissolved, flocculent, ragged, and hanging in shreds. The opening is much less abrupt than that found when a corrosive poison has perforated the stomach.

It is not clear what are all the circumstances necessary for the production of this extreme solution. There can be no doubt that a healthy digestive process in operation just before death is the most favorable for it, and thus it is that animals killed with food in their stomachs are so often found to have the organ dissolved; and for the same reason it is in persons who have died suddenly during digestion that the stomach is most affected. Constantly, however, as we are meeting with persons dying under these circumstances, it has not been especially in them that perforation has been met with, for during last year the only two cases observed were one in a child who died of brain disease, and another in a lad, with albuminuria; in another more recent case there was disease of the pons Varolii. It has been thought that the ordinary processes of decomposition have had their share in its production; but this does not seem so, for now and then it falls to our lot to examine bodies frightfully decomposed, but in none such have we found perforation. It has been thought, too, that warm weather has influenced it; but this notion had arisen probably from the supposition that decomposition is more rapid in warm weather than cold, which is by no means the case; the condition of atmosphere which most favours decomposition is moistness, and thus a moist cold day in winter is more favorable to the general disintegration of the body than a warm dry day in summer. This we have observed on repeated occasions. It is clear that warmth would favour solution, but then it is manifest some other cause must be in operation to determine it in one case more than another; and it may be due, perhaps, to the formation of a morbid excess of gastric juice; this, however, has yet to be proved.

**Idiopathic Softening.**—We mention this in connection with post-mortem solution, because it is stated by some writers to be a frequent pathological condition in children. It is called *gelatiniform softening* of the stomach, and described by some Continental authors as productive of certain symptoms, and leading to a fatal issue. Of this condition we know nothing, and in fact feel convinced there is no such disease as gelatinous softening, all that we have observed being cadaveric.

**Morbid Contents.**—We should always examine the contents of the stomach after death, to see whether they consist of food or merely mucus, and to observe their colour; if brown or black, it may be owing to blood or some medicine administered; blood may come from the nose, chest, &c., or from a fractured skull. Chemical tests will detect iron, as the microscope will blood.

Extraneous matters are sometimes found in the stomach, as a mass of hair; and in one specimen on our shelves the organ was found to contain the parts of several knives. The stomach was much enlarged, and its walls thickened. It came from the body of a sailor who was known as the knife eater. Mr Poland collected together a most remarkable list of cases where foreign bodies had been found in the stomach. His paper is in vol. ix, series 3, of the 'Guy's Hosp. Rep.'

The *quantity* present is sometimes significant. Thus we have found two pints of food in the paralytically dilated stomach of a fever case.

Some think that we may find cancerous growth in vomited matters. This is just possible, but the most interesting object met with is one of modern discovery—the *Sarcina ventriculi*, a vegetable fungus discovered by Goodsir. The sarcinæ are at once recognised in the contents of the stomach by their cubical figure, each face being made up of four small cubes, and thus resembling a pack with a cord round it, whence the name sarcina. Many of these may be united together, forming large bundles, and occasionally they have a greenish cast: they are found in those cases, especially of obstruction, where the food ferments.

We must notice the occasional occurrence of parasites which appear to correspond to the *bots* with which the stomachs of cattle are affected, and which proceed from a fly called the *Œstrus bovis.* A certain doubt must attach to all kinds of maggots and other wonders shown by patients; but some of the cases are so well authenticated that there can be little doubt that the œstrus does, very occasionally, affect man.

*Air.*—We not uncommonly find gas in the form of bubbles beneath the mucous membrane. In all such cases which we have seen it has arisen from decomposition: a true emphysema from extravasation of air we have never witnessed.

## INTESTINE

**Malformation.**—This shows itself chiefly in two places. First, at the seat of the fœtal vitelline duct, which originally passes through the fœtal umbilicus to join the umbilical vesicle. The persistence of this duct appears to give rise to a diverticulum from the ileum, which is frequently met with in the post-mortem room, and of which we have several specimens; such diverticula generally jut from the lower end of the ileum, about two or three feet or more above the cæcum; nearly always on the free border of the bowel. They are of various lengths; we may find them as pouches ranging from the smallest size to portions several inches long, like the cæcal tubes in birds. Their structure is exactly like that of the neighbouring part of the ileum, and they partake in its morbid processes, so that typhoid ulcers may be found in them. The diverticulum may extend through the umbilicus of the fœtus, and so be divided when the cord is cut; it may then be attached or not to the umbilical opening. Under these circumstances a plastic operation may close the bowel, but afterwards strangulation by the band thus formed has been known to occur.

Several cases of fatal obstruction have been found to be due to a narrowing of the ileum itself at the spot where the diverticulum is given off. In one case under Mr. Davies-Colley of an infant there was almost complete occlusion at this spot caused by the presence of a septum within the bowel. It was exactly nine inches above the cæcum.

The second principal seat of malformation is the end of the bowel. The simplest and most important condition of the kind here is absence of the lower end of the rectum—*atresia ani*—requiring operation to establish an anus. A greater length of colon may be wanting, so that to reach the gut operation in the loin or groin is required. In much rarer instances the small intestine may be blind at one or many points; such badly malformed cases are, fortunately, unviable; as, also, fortunately, are most of those conditions where a cloaca is formed by non-development of the divisions between the genital, urinary, and fæcal passages. In these, various combinations of the rectum, vagina, and ectopic bladder are met with. In cases of yet less complete development the ileum may open upon the ectopic bladder. A complete occlusion of the duodenum has occasionally been met with in the infant, as in a specimen on our shelves, and about which full particulars are to be found in the catalogue.

**Malposition** of the intestines, either as herniæ or as displacements within the abdominal cavity, are common. When the liver is flabby it sometimes falls back, and the colon rolls over its edge, separating it from the anterior abdominal wall, making the liver appear much smaller to percussion than it really is. Internal displacements may arise through adhesions of the intestines in early life to other parts, so that they are prevented from taking their natural places during the growth of the body; when the position is, as in many cases, greatly altered, it is tolerably certain that the union must have occurred in fœtal life; and this becomes another illustration of what we have said, when speaking of malformation of the heart, that such conditions often arise from disease *in utero.* In the present instance we can speak with more certainty of the occurrence of such inflammation, for we have several times met with peritonitis, both recent and old, in the new-born child, especially in syphilitic infants. Adhesions occurring in extra-uterine life no doubt alter the position of parts to a certain extent; and thus we find the colon drawn down to the pelvis, or the sigmoid flexure carried upwards, &c. Such displaced parts may twist on themselves; or else form loops, under which coils of intestine may become strangulated.

But twists of the bowel often occur without any preceding adhesions. Such a twist, however arising, is called *volvulus.* When this accident has come under our observation, there has evidently been unnatural length of the mesocolon. It usually occurs in elderly males, whose time of life would show that the lengthening of the mesocolon is an acquired change. Volvulus happens most frequently at the cæcum or sigmoid flexure, but it has occurred in other parts of the colon, and even in the small intestine. Sometimes the canal twists on its axis; sometimes a coil twists on its root. A coil so twisted may dilate to an immense size, while it becomes very dark purple in colour. Thus a twisted sigmoid reached the under surface of the diaphragm, as did a twisted cæcum. Sometimes the twist is more complex—one coil is wrapped around another coil; when this is so, there is generally some awkward adhesion of the coils together causing the twist. We have already mentioned the curious twists to which the bowel is subjected by contraction of peritoneal cancer. This may cause fatal ileus, generally very gradual in its approach, and perhaps hidden amongst other symptoms. The distended part may often atrophy, ulcerate, and rupture.

*Hernia.*—Another kind of malposition forms the great and important class of intestinal hernias, where the bowel escapes from the abdominal cavity. The more ordinary kinds of hernia, namely, *inguinal*, *femoral*, and *umbilical*, are so fully treated of by the surgeon that we shall not dwell upon them, but will mention only those rarer kinds which are usually discovered at post-mortem examinations as causes of obscure abdominal obstruction during life. We may observe that when the bowel in hernia sloughs open, the breach is along the line of stricture; but if it be burst in taxis the rent may be in the prominent part; such accidents may produce fæcal abscess. Among the rarer places where hernia forms is the *obturator* foramen. Here, again, it may occur without any pain or tumour. Another is the *ischiadic notch* in the course of the sciatic nerve; this is a very rare occurrence. A hernia rarely forms behind and beside the anus, through the hinder part of the perinæum, *perinœal* hernia; or into the labia pudendi, *pudendal* hernia.

*Ventral* hernia is like umbilical, but passes through some casual weak spot which allows an opening in the fascial coats of the abdomen, in the middle line or to one side of it. Besides these forms of hernia the intestine may force its way outwards by bulging in the wall of the vagina, and protruding from the vulva, *vaginal* hernia; or in a similar way through the rectum, *rectal* hernia, protruding through the anus.

*Diaphragmatic* hernia is generally caused by grave injuries bursting the diaphragm, so that the patient is killed quickly by the accident; but cases are recorded wherein the patient has survived such an accident for a long while. There may be no gastric symptoms, as in a case recently under our care. Other examples are formed more slowly through yielding of the weak portions of the diaphragm, and some are congenital; these are, however, rare. Another interesting kind of hernia is known as *retroperitoneal;* it is very unfrequent. The few cases which have occurred have been collected by Dr. Pye-Smith, and will be found in vol. xvi of the ‘Guy's Hospital Reports.’ The intestine pushes down behind the left mesocolon, passing in above, and down behind the inferior mesenteric artery, into the lax subperitoneal tissue; or, in one case, behind the superior mesenteric. In this way a large downward pouch may hold nearly all the small intestine. We have once seen an upwardly directed pouch behind the right mesocolon turning in below the mesenteric artery. We may remark that a hernial sac may be returned into a subperitoneal position with the stricture unrelieved. This is described in surgical works under the heading "*reduction en masse.*"

**Incarceration and Strangulation.**—This is the case in which the bowel becomes imprisoned and strangulated by a band around it; and is a relatively frequent cause of death. Such a band is generally either a diverticulum of the ileum, or else the cæcal appendix. The latter is excessively variable in its position, sometimes being altogether behind the peritoneum, and sometimes free in its whole length. We have known it to form the only contents of a femoral hernia, and we have found it touching the gall-bladder; but it may be attached at its end and free in its middle, so as to form a loop always ready to allow of the entrance beneath it of a coil of small intestine. A diverticulum of the ileum attached at its end becomes a similar snare. In some cases this diverticulum appears as a solid cord, representing, as it were, the vessels that once accompanied the vitelline duct, but sometimes such cords are too high for the vitelline duct. Besides these, the more usual causes of external constriction, we have seen the colon crossed at its splenic flexure by a simple thickening of the peritoneum and so strangulated; or there was a hole in the omentum, a mere gap, through which only one knuckle of ileum had got; or in the mesentery a similar hole, or a pouch formed within it (such a pouch constitutes one sort of extra-peritoneal hernia); or the omentum attached to an ovarian tumour, or to an extra-uterine fœtation, strangulating the intestine; or an adhesion caused by tubercular ulceration within the bowel; or a loop produced by adhesion about the appendages of the broad ligament of the uterus. Any such causes of external constriction may catch the bowel, especially the small intestine, and cause perhaps slower obstruction, but far more generally a quick and fatal form of ileus.

**Intussusception.**—This disease, judging at least from our own experience, is not nearly so common in adults as is generally supposed; and as a cause of fatal obstruction in the bowel is by far less frequent than the bands of peritoneal adhesions already mentioned. Before morbid anatomy was as much cultivated as at present, it was commonly assumed in cases of internal strangulation, where symptoms were sudden and speedily fatal, that such an introversion of the intestine must have occurred; we should say, however, that rapidity of symptoms rather militates against the probability of intussusception, seeing that the process of its formation is mostly slow and the impediment at first partial. We have seen but few cases of intussusception in the adult, and it is remarkable that these have frequently been occasioned by growths in the wall of the bowel, especially in the colon. In one of our cases of intussusception of the ileum, in a girl of seventeen, the starting-point was at a diverticulum of the ileum. Another in the ileum, examined by Dr. Cayley, began in cancer there, a rare disease, indeed, in that situation. In these cases the obstruction is generally never complete, and death does not occur for some weeks. In infants, however, the affection may rapidly develop, and be speedily fatal. It is only where the introversion is considerable that symptoms occur; it is highly probable that a slight slipping in of one portion of bowel into another is frequently taking place, if we judge from what is met with on the post-mortem table; in children especially, who have died from cerebral disease, the intestines are found contracted, and often invaginated in several places. These are readily pulled out. The occurrence in this class of cases naturally suggests a nervous influence productive of the condition; it is mostly found in the small intestine. The graver intussusceptions are also more frequent in the small intestine. Thus, among twenty-nine cases of separation of the invaginated part collected by Dr. Peacock, the piece of bowel was found to be ileum in twenty-two examples. The common seat is at the end of the ileum, which enters the cæcum.

The part that first enters continues to keep the most advanced position of the whole intruded mass in its further progress. We can easily see how the passage is to a certain extent pervious, by attempting to imitate the invagination on the finger of a glove: if we simply force one part into another, and then cut off the top so as to leave the channel open, it will have some resemblance to an intussusception. Students are often at the moment puzzled, when asked to enumerate the layers met with in a section of an intestine so invaginated; but it only requires a moment's consideration to know their relations.

There are three portions of tube including and included. The inside one joining the middle at the most advanced or deepest part of the invagination; the middle one joining the external at the least advanced part; between the inside tube and the middle one is a recess of peritoneal surface in the direction of the course of the intussusception; this recess may contain some blood and lymph; between the middle tube and the outside one is a recess of the mucous surface closed in the reverse direction. If now we examine these three tubes of bowel we find that they suffer in very different degrees and always quite characteristically; the part that suffers least is the outermost tube, while the part that suffers most is the middle tube, especially at its junction with the innermost. This junction is the original starting-point of the intussusception, and if a cancerous or other tumour caused it, it is here we find that tumour. If there is no cancer we might often be led to think one present, through the surprising swelling of the bowel wall at this bend. We have measured the bowel three quarters of an inch thick here; its mucous surface is intensely congested and eroded, or hangs in sloughy shreds, hence the bleeding which is so characteristic of invagination of the intestine.

To see the amount of mischief thus produced we must open the containing bowel, or sheath, and thus expose the included portion within; we then discover that the example of the invaginated finger of the glove does not perfectly hold good, nor, indeed, the ideal or diagrammatic drawings of an intussusception, for in these the tube is supposed to be simple and free, but the intestine has a mesentery attached, which acts a very important part in the process; it is this membrane which prevents intussusception from more frequently occurring, but if once intussusception has taken place, the mesentery attached to one border of the bowel pulls on it so as to make the mouth of the enclosed part turn to the side to which the mesentery is attached, and presses it against the side so as almost to close the orifice. Thus, when we open the outer intestine, or sheath, we do not find the opening of the bowel at the extreme end of the intensely congested mass within, but turned to one side of it. This dragging of course obstructs the vessels of the mesentery, and, consequently, this structure is found of a dark colour, and sometimes indurated from blood effused within it. The seat of stricture is always at the junction of the middle with the outer tube, and all the included part is strangulated; it becomes of a dark colour and, perhaps, gangrenous, so that it tends to become separated; and during this process the opposed serous surfaces of the inner and middle tubes, at the point where the one portion of bowel enters into the other, are becoming united by lymph which is formed through inflammation. This is the process of cure; but during the sphacelation and separation death often occurs through an ulceration of the intestine at the point of junction, or an extension of the inflammation through to the peritoneum. Recovery is sometimes complete; this is, however, rare. The patient generally suffers afterwards with signs of abdominal obstruction. Thus, we have met with two instances of fatal annular stricture of the ileum with puckering of the mesentery at the spot affected, all no doubt due to former intussusception, though no history of this was obtained; others have recorded similar cases. In explaining the absence of history we must note that the piece of bowel passed may be small, and recovery is often then remarkably rapid. The piece of bowel passed may vary from two to forty inches in length; and in one remarkable case, quoted by Dr. Peacock, eight pieces, passed in three years, amounting in all to no less than twelve feet.

A specimen in the Museum came from a child aged eight years, who, after eleven days of obstruction, passed the cæcum and ascending colon. She then rapidly recovered.

A very remarkable case occurred in the private practice of Dr. Goodhart, where the commencement of the jejunum became invaginated, so as to form a tumour in the abdomen eighteen inches long.

The patient was a young girl, and the swelling had been recognised as productive of her symptoms for twenty-one months ('Clin. Soc. Trans.,' xix, 146).

The Museum contains a remarkable specimen of a large portion of mucous membrane, showing valvulæ conniventes passed *per anum.* Its passage was preceded by pain in the abdomen and hæmorrhage, and Sir W. Gull believed, therefore, the sloughing was due to thrombosis of the mesenteric vein. This opinion was supported by a specimen in the Museum presented by Dr. Fagge, where death occurred from occlusion of the mesenteric vein and sloughing of the mucous membrane.

*Other causes of obstruction.*—We may here mention the other causes of intestinal obstruction. Among them are those seated in the wall of the bowel. Thus simple, tuberculous, syphilitic, or especially cancerous ulceration may give rise to *contraction* of the bowel, or the cicatrix left by former ulcers, or by the separation of an intussusception, may for years produce chronic intestinal trouble ending in ileus. Stricture of the rectum may be produced also by implication of the gut in pelvic abscesses connected with the internal genitalia; such abscesses may open into the rectum, and by contraction may twist or bend it so as to cause chronic obstruction. We have already mentioned other causes under "Malformation."

*Prolapsus ani* is a condition resembling intussusception, only that the external tube is wanting. If the prolapsus is small, it is of no great importance; but if great, a strangulation of it similar to that which causes gangrene of an invaginated bowel may occur, and although gangrene is rare, yet the extruded part may become congested and inflamed, swelling to a large size. The prolapsus of congested mucous membrane only, may be mistaken for a prolapsus of the whole bowel.

Lastly, yet another kind of obstructive agency is found in *concretions* stopping up the canal; usually large gall-stones, which have made their way into the duodenum by ulceration from the adherent gall-bladder, or which may even have passed down the dilated bile-duct. These may be stopped in the narrow portion of the ileum and cause fatal ileus—we have here several such preparations; or else intestinal concretions may act in the same way, such concretions being composed of hair or woody fibre, magnesia, &c. In some rare cases ileus is produced by enormous accumulations of fæces in the lower end of the colon. But the danger from these accumulations is rather that they tend to produce ulceration, and perhaps perforation, of the bowel.

**Dilatation.**—Enlargement and distension of the intestine, especially of the colon, is found above the seat of chronic stricture, sometimes reaching an enormous extent. The muscular coat at the same time is hypertrophied, so as to be twice to four times as thick as natural. The effects of stricture on the dilated intestine may be very severe, especially if purges be given. Thus, we have sometimes seen the peritoneal coat and the muscular coat cracked in several places, when the colon has contained eight pounds of fluid above a stricture at the sigmoid. This giving way occurred at the cæcum, and not near the sigmoid. Also we have seen the whole thickness sloughed through over several square inches; or the mucous surface may be ulcerated more or less severely; this also may happen at some distance above the stricture. We have met with some cases in which the intestines were punctured when distended with gas, and no ill result followed; but twice peritonitis occurred in similar cases.

Sometimes we meet with cæcal pouches from the colon. These are hernial protrusions of the mucous membrane outwards through the muscular coats. They appear to be innocent; sometimes they are numerous; they occasionally contain fæcal matter, and might undergo ulceration and perforation, but we do not know of this as having occurred. In one case pouches in the small intestine protruded through the layers of the mesentery. These pouches and acquired diverticula must be distinguished from congenital diverticula.

*Primary dilatation.*—We sometimes meet with enormous dilatation of the large intestine without any obvious stricture to have caused it. Of this kind is a specimen which came from a young girl under Dr. Goodhart, in which the colon was immensely distended and the walls of the intestine extremely thickened. Several cases of the kind have been taken to the Societies where the distension was so great as to cause difficulty of breathing and death. In the absence of any mechanical cause it has been regarded as due to paralysis. We have already mentioned the analogous case of primary dilatation of stomach.

**Injuries.**—These are mostly fatal, from the fact of some of the intestinal contents being extravasated into the peritoneum, and setting up inflammation; otherwise wounds of the bowel may be healed. Thus, if the wounded intestine should protrude, or can be secured and stitched up, a cure may follow, the threads finding their way ultimately into the canal. Generally the mucous membrane protrudes at a wounded spot, through retraction of the muscles; but we have seen the reverse occur, the seat of puncture dipping in towards the channel of the bowel, and the peritoneal surfaces around the puncture being thus brought in contact, so as to cohere and favour a cure; not, however, enough to prevent fatal peritonitis. Many of the injuries to the intestines which we witness occur from falls, or from wheels of vehicles passing over the abdomen. The parts most exposed in run-over cases are the duodenum and jejunum; for the ileum, in the lower part of the abdomen, is shielded by the pelvic bones, while the lumbar spine rising forwards, without ribs or pelvis to take the pressure, offers a hard surface upon which any unfortunate piece of bowel may be crushed or dragged away by the wheel. Often also the intestine is ruptured by the kick of a horse, or by any other sharp blow, or a fall; and it is very important to remember that such a fatal injury may occur without any external bruise. The intestine may be entirely torn across; the mucous membrane then protrudes, while the peritoneum is simply divided, and the muscle retracts. Sometimes the bowel is bruised and inflamed without bursting, and peritonitis is set up.

**Congestion.**—This is generally of a passive kind, and associated with a similar condition of the stomach, as already mentioned, among the consequences of heart disease, &c.; engorgement of the portal system occurs and thus congestion of the mucous membrane of the stomach and intestines. The congestion is, however, more direct in primary hepatic obstruction, as cirrhosis, &c. Owing to the resistance to the current in the portal vein, the serum escapes on the peritoneal surface, constituting ascites; while on the mucous surface the blood escapes entire, and so may be found in the intestine. If there is no bleeding, the mucous membrane is highly congested. We have just now alluded to mechanical impediment to the flow of blood as producing bleeding in intussusception of the bowel where the congestion is excessive. We must not be misled by congestion from mere accidental dependence of the coils. The blood settling down to the deepest part of the coil produces an unequal congestion that surprises beginners. We have seen great congestion in pyæmia, and this has suggested a cause in a morbid state of the blood itself. We know that in septicæmia produced by experimental injections of putrilage into dogs' veins, the chief symptom is diarrhœa. When in slight degree, congestion may perhaps arise from an irritant in the shape of food or medicine, the redness is then observed more on the rugæ; if there be any large quantity of mucus or lymph adherent to the prominent parts, we must consider that the hyperæmia is inflammatory.

**Œdema.**—In cases of dropsy we sometimes find the whole structure of the bowel in an extreme condition of œdema, so that its weight is very much increased, and it has a gelatinous appearance. This condition must greatly impede the functions of the bowel, but we have not been able to trace any special symptoms to it. It appears sometimes to be increased by irritants, as croton oil. We have thought that it sometimes has obstructed the mouth of the bile-duct, causing jaundice.

In persons who die during digestion of a full meal, we may see the villi whitened by chylous fluid within, also the lacteals traced out plainly as white lines.

**Lardaceous Disease.**—This is rather frequent in the alimentary canal; it produces a state of the mucous membrane more easily recognised than described. The surface looks like wet wash-leather; if it be cleaned and iodine applied, it becomes of a deep walnut colour.

Microscopic examination shows the minute arteries chiefly affected; but the amyloid matter extends from these into the tissue around. Peyer's patches generally are less implicated, which is remarkable when we remember their structural identity with the Malpighian bodies of the spleen, for these are always first affected in that organ. If we are searching for lardaceous disease, we choose a Peyer's patch and the part around for application of the iodine, and the contrast of effect is at once seen. Death by diarrhœa is common in these cases, for there is liability to catarrh, or even severe enteritis, which may lead to sloughing.

**Inflammation of the Intestines.**—The term enteritis, or inflammation of the small intestine, has generally been used to denote certain symptoms of derangement of the bowels, with a more vague signification than would be given it by the morbid anatomist.

*General enteritis.*—A general acute inflammation of the whole interior of the intestinal canal, without any obvious reason, as a special affection to which we can give the name idiopathic enteritis, is certainly not common, if it exist at all.

A general *acute catarrhal* irritation of the alimentary canal occurs under the same conditions as the quite similar catarrh of the stomach, *e. g.* in pneumonia, fevers, croup, &c.; it causes a pinkness or redness of the mucous surface, and an increase in the quantity of mucus on the surface, which mucus becomes also semiopaque.

A *chronic catarrhal* inflammation is shown by enlargement of the glands, and by darkening of the surface to a dusky, slaty, or even to a black colour (this black colour disappears in spirit-kept preparations). There is an excess of mucus on the surface. In some states of chronic irritation, the solitary and agminated follicles become blackened, producing an appearance well compared to that of a shaved beard. Often in the post-mortem room we cannot connect these appearances with definite symptoms, but there is no doubt that they belong to old chronic enteritis of a degree milder than would attract remark among the symptoms of the fatal illness. They most frequently occur in cirrhosis of the liver, cardiac disease, or other causes of portal congestion.

General enteritis, distinguished as "toxic," may be set up by irritant poisons, and therefore in suspicious cases the whole canal should be carefully examined. We usually find more severe effects in the large than in the small intestine. And, indeed, it is remarkable how the poison may pass down the small intestine without affecting it, and then set up violent inflammation in the large. This has especially been the case in two or three instances of poisoning by perchloride of mercury which we have seen, where the small intestine had escaped with impunity, but the large had even had its mucous membrane destroyed. On the other hand, in a case of phosphorus poisoning, the small intestine was more affected than the colon.

*Local enteritis.*—The long tract of the intestine is liable to be affected with many changes that are included under the wide term inflammation; also in the variously constructed parts of the canal some inflammations elect one seat and some another; so that local names are given to the inflammations of special seats, such as *duodenitis, ileitis, colitis, typhlitis, proctitis,* &c. Besides these varieties as to the *position* of simple inflammations, there are special *kinds* of inflammation, such as *tubercular, typhoid, dysenteric,* &c., but of these we shall speak afterwards.

If the duodenum is especially affected, the disease is called *duodenitis.* The changes which no doubt are constantly arising in the stomach from improper food are propagated to the duodedum, and it is even thought that some forms of dyspepsia are due to its inflammation, and that a continuation of such affections up the bile-duct produces jaundice and other affections of the liver. The duodenum may certainly be found highly congested in the same case where the stomach suffers. In poisoning, too, the duodenum shares the inflammation. *Ulcers* are met with in this part of the intestine relatively often when they exist nowhere else. On many occasions we have met with them. These ulcers correspond in their general character with gastric ulcer, and, like these, sometimes give rise to perforation and fatal peritonitis. As to their cause little is known; but these ulcers occur in the upper part of the duodenum, and this part is in contact with acid contents, like the stomach itself; such ulcers do not often occur below the bile-duct, which supplies alkali to the intestine. The most interesting fact respecting ulceration here is the observation of Mr. Curling, that it often is met with in cases of burn; of this association he has cited numerous examples. We have met with a few such ulcers, which we think are not so common as he suggested; some of these burn-ulcers have been also found elsewhere in the alimentary canal. We have seen one example in the stomach; and in one other of our cases, where a duodenal ulcer after a burn was fatal by hæmorrhage from the widely open pancreatico-duodenal artery, there was a deep eroded ulcer on the back of the tongue. In another case the ulcer opened into the portal vein. Some of these duodenal ulcers from burn go on to perforation, and so kill by peritonitis.

Gall-stones sometimes ulcerate their way into the duodenum from an adherent gall-bladder.

Post-mortem solution and perforation sometimes occur in the duodenum. We meet with great difference as regards the size of Brunner's glands in the duodenum; sometimes they are scarcely visible, at other times very large and prominent, as we see in a drawing which was taken from a case of cholera. Sometimes one or more grow so large that they are described as "accessory pancreas."

Occasionally a *general ulceration* may be found existing through the whole of the small intestine, in the form of numerous ulcers with raised vascular edges, and without any disease in any other part of the body. These ulcers do not run in the course of the intestine, but their greatest length is from side to side, so as to entirely encompass the circumference of the bowel; if they do not quite meet, the incompleteness is on the side of attachment of the mesentery. Such cases are very rare, and when met with their pathology is not very clear, but the disease seems to be primary.

The *jejunum* is almost free from primary disease, but we have twice met with clean-cut perforating ulcers of the jejunum similar to "punched ulcer" of the stomach, there being no other disease present in the body, and the course of the cases being that of sudden and acute disease. Ulcers may be due to ileus from obstruction lower down.

We have met several times with severe local acute ileitis in the shape of a thickening of the whole of the coats, including the valvulæ conniventes, which stood out stiffly, while the whole wall was thick with inflammatory lymph; the microscope showing a general charging of the whole tissue with pyoid corpuscles. This condition was found in a circumscribed patch of from six inches to two or three feet. Once the disease was uncomplicated; once a fatal condition in diabetes; once it arose in Bright's disease; and again, once in a yet severer form, going on to sloughing of the affected part, in the lardaceous intestine of an old syphilitic subject. Dr. Dickinson has also found it in Bright's disease, and a very similar condition in those who have died from anthrax. In scarlatina Peyer's glands are often much enlarged, and in some other forms of specific disease. In various forms of malarial disease Dr. Vandyke Carter has found ulceration of Peyer's patches. In long-protracted diarrhœa some ulceration may be always looked for, except in children, where the diarrhœa or gastro-enteritis is often more an irritative effect of unsuitable food than a proper inflammation. In fatal cases of this kind we generally fail to discover any other appearance than an excessive enlargement of the intestinal glands; thus we may see the duodenal and Peyerian glands enlarged, and even more the solitary glands, both in the small and large intestine; these are seen as small bodies projecting from the mucous membrane. We should remember, however, that these glands, as well as the mesenteric, are comparatively larger in children than in adults; and thus we have heard students inquire if such have not been enlarged when we judged them to be only natural. We sometimes meet with simple follicular abscesses of the submucous coat, of a size from that of a lentil to that of a pea. These are usually found in scrofulous subjects, and then are seen as little sacs of pus; they are very different from the follicular *ulcers* which are associated with miliary tubercle in phthisis. We have not ourselves found tubercles secondarily developed around these follicular abscesses, though such an occurrence has been described.

*Typhlitis or inflammation of cæcum and appendix.*—This is frequent and important, so as to require our special attention. When an inflammatory process has been confined to the coats of the gut itself, the term *cæcitis* or *typhlitis* has been given; and when the cellular tissue around is especially involved, *perityphlitis.* Systematic writers speak of inflammation of the *cœcum proper*, inflammation of the *appendix*, and inflammation of the *pericœcal tissue.* It is not clear, however, that any one particular form of disease is here intended by those who make use of the expression typhlitis. The cases to which this name is given frequently occur clinically, and recover; but when disease in the same region with similar characters proves fatal, we find usually some prior morbid process in the appendix rather than in the cæcum itself. The appendix, again, may be the subject of ulceration in fever, phthisis, &c., as we have just seen; it may have various morbid secretions occurring in it, or concretions, or even foreign bodies lodging in it, which shall light up inflammation in the surrounding parts. The suddenness of the attacks of cæcitis, and the local peritonitis following, even in the large number of cases which recover, all point to the appendix as being the most frequent cause. Inflammations of the cæcum itself do occur, and sometimes are apparently caused by the continued lodgment of hard fæces in this part of the intestine. Such inflammation may, by ulcerating the mucous membrane, lead to perforation and local peritonitis, forming a fæcal abscess in the neighbourhood, which may discharge inwards, but we believe that this is comparatively rare. It must be remembered that the appendix may be found in various positions; its more usual one is behind the ileum and pointing upwards, and this may determine also the position of the abscess.

In some cases it does not appear that an actual perforation has occurred; the inflammation may implicate the adjacent parts in the inflammatory process by contiguity. In those which recover, the inflammatory symptoms slowly subside, or an external suppuration may follow, and the cause remain unknown; and in fatal cases so general a peritonitis may have occurred, or so much suppuration around the cæcum, that it is then difficult to arrive at the exact nature of the complaint; thus we may find the appendix sloughing or gangrenous, as if some foreign matter had been discharged from it, and yet be unable to discover it. Foreign bodies of small size may collect in the appendix, as in a specimen taken from a woman who died of hernia, and who, to obtain a passage through the bowels, swallowed a quantity of shot; these we see have lodged in the appendix. We believe, then, that many of the cases of disease supposed to affect the cæcum, arise rather from a chronic morbid process in the appendix. If we are in the habit of examining this process we may often find a morbid secretion within it (the mucous membrane possessing glands), a thick mucus, and, sometimes, a material like wax, which appears quite peculiar to it. The appendix is found rigid, and on opening it is seen to contain a semi-translucent matter like wax, which on section often consists of concentric layers. This is sometimes of a brown colour and hard, and at other times earthy matter is combined with it, until a perfect stony concretion or calculus is formed; this has a disposition to ulcerate its way through. Frequently, in cases that die after a sudden attack of peritonitis, this is found due to such a calculus, which had made its way through the appendix. Such cases are more common in young persons, especially of the male sex, which amount, indeed, to four fifths of all the number. In some cases the exterior of the concretion is earthy, and the centre soft and partly feculent. We read in books that cherry-stones, and especially date-stones, have often accidentally lodged in the appendix, and given rise to the subsequent mischief. We have never yet met with these bodies, but the hard brown concretions in the vermiform process very much resemble date-stones, so that one cannot help a suspicion that mistakes have often been made as to their character.

*Inflammation of large intestine.*—The term colitis is sometimes used as though synonymous with dysentery. Our usual language has been too indefinite, nay, incorrect, in speaking of all affections of the large intestine as dysenteric; for the true dysenteric process, although in many features like simple ulcerative colitis, is a disease having certain definite characters.

There is quite as much reason to regard febrile epidemic dysentery as a disease distinct from simple ulcerative colitis, as there is to regard febrile epidemic diphtheria as a disease distinct from idiopathic croup. We have already seen how imperfect are the merely anatomical means of distinction between diphtheria and croup; and there is equal difficulty in distinguishing dysentery from simple colitis by anatomical characters. Dysentery, in fact, produces a variety of colitis distinguished from others by a special course, and by clinical phenomena; though when the dysenteric fever has passed off, the state of the colon which is left is much the same as that of simple ulceration.

Cases of true idiopathic colitis are rare, and stand, as we have said, in the same relation to true dysentery as croup does to diphtheria, from the absence of those peculiar features which give dysentery the characters of a specific fever. For example, we have seen a case attended by discharge of mucus and blood where, after death, the whole internal surface of the colon presented a highly vascular, soft, red surface, covered with tenacious mucus or adherent lymph, and here and there showing a few minute points of ulceration; the coats, also, were much swollen by exudation into the mucous and submucous tissues. Bristowe has observed an inflammation of the large intestine or a colitis not unfrequently in fatal pneumonia. In other examples there has been extensive ulceration, commencing in the follicles, and spreading from them to destroy the tissue around, thus producing a ragged ulcerated surface, which is so far identical with the effects of the specific fever dysentery, that the distinction between the two conditions rests on the characters of the accompanying constitutional disturbance, just as scarlatina is known from simple cynanche by the addition of its proper fever to the swelling of the tonsils.

Ulceration of the large intestine is one of the most common post-mortem appearances we meet with; in persons long ill with various visceral complaints we often find large ulcers in various parts of the large bowel, but more especially in the cæcum, ascending colon, and sigmoid flexure. These are mostly of a chronic character, with raised indurated edges ; some spreading while others are healing. They probably have the same pathology as many ulcers on the leg connected with a retardation of venous circulation. These ulcers have their length in the direction of the transverse course of the bowel, passing around its circumference. In cases of phthisis and tubercular disease, it is not uncommon to find such ulcers, and not, apparently, having a tuberculous character. In some cases ulceration of the bowel constitutes the main disease and must be regarded as primary, as in a man living near here whose colon was ulcerated and cæcum perforated.

The rectum is very liable to be perforated by pelvic abscesses ; also the cæcum by lumbar abscesses occasionally.

*Pellicular or diphtheritic colitis.*—This is the kind of inflammation where the secretion is firm, and forms an adherent layer on the surface, but has no necessary connection with the specific disease diphtheria. It is not unfrequently met with in the colon. On opening the large intestine, its whole inner surface appears of a grey colour, the mucous membrane being hidden by a firmly adherent deposit upon it ; this can be stripped off with difficulty. It is found to be very tough, and its surface is granular ; the mucous membrane beneath for the most part looks healthy, but in some places very minute ulcerations are present, occupying apparently the sites of the follicles, and in other parts it is highly congested. In these two specimens the lower end of the ileum was also involved, but only in patches ; a stream of water flowing upon these failed to move the adherent lymph. A section shows the walls somewhat thickened, and although this may be due to the contraction of the gut, it is evident that in many cases there is an effusion of fibrin into the submucous tissue. We have seen this diphtheritic colitis occur in two or three instances as an idiopathic and fatal form of disease. We have several times met with it along with a similar inflammation of the stomach ; also in Bright's disease. We have met with it, too, in diphtheria of the fauces, and a similar experience has happened to others. In these acute cases the adventitious matter could not be detached as a distinct membrane ; but occasionally we meet with chronic forms of pellicular inflammation of the large intestine such that *casts* of the intestine may be passed as well as long pieces of mucus, which are often mistaken by the patient for worms. These long pieces consist mostly of mucus containing cells. Occasionally, if placed in water, they will be found to be hollow, and to correspond to the part of the gut from which they have exuded, constituting indeed a mould of the intestine, the microscope detecting on them the marks of the follicles, and of the arrangement of the mucous membrane. The patients who suffer from this condition generally exhibit great mental depression, and are amongst the most intractable of valetudinarian melancholics.

**Fistula in Ano.**—We may here mention the anatomical characters of *fistulæ in ano.* There is still a doubt whether these begin first in ulcers which perforate outwards, or in abscesses that burst inwards; probably some arise in each of these ways. The history of pain at the commencement and sudden relief will signify the latter origin. We have traced some clearly to the suppuration of inflamed hæmorrhoidal veins. The fistula is found as a callous passage in the fatty tissue about, or in, the wall of the anal part of the rectum. By far the larger number of fistulæ do not open higher than an inch and a half within the bowel, and these generally have their external orifices farther from the anal aperture. But those that run high often open near its margin. The internal communication may be at the top of the fistulous channel, but sometimes there is yet a sac above this opening, and sometimes the fistula forms branches and has several openings into the bowel; the tissues about the anus are then dense and vascular. Fistulæ are more frequent in phthisical patients. We have twice seen tubercles in their wall under these circumstances. Severe accidents rarely arise from operations on fistulæ, but we have seen once pyæmic abscess of the brain follow such an operation.

**Enteric Fever.**—Among the specific inflammations of the intestines there is none more interesting and important than the enteritis of typhoid or enteric fever. This shows itself in a peculiar affection of the solitary and agminated follicles, especially of the lower end of the ileum.

This state of the glands is the pathognomonic feature of enteric fever; and although there are other dangers in the fever, yet the consequences of these changes in the bowel, as perforation or hæmorrhage, bring about a large proportion of the deaths from this disease. The swelling of Peyer's patches and the solitary glands is more severe in proportion to their nearness to the cæcum, so that we often find several Peyer's patches in the upper ileum or jejunum unaffected while those near the cæcum are destroyed. Rarely a higher patch is worse than a lower.

The appearance of the glands depends on the stage of the disease at which the examination is made; but in average cases, as of death early in the fourth week, we find close to the cæcum some patches destroyed and almost entirely removed, and higher up some swollen and partially destroyed; and higher still we find the disease limited to particular follicles in a patch, so that the latter has only a small spot or two of the affection. The history of the changes in the solitary and agminated glands is shortly this. Under the influence of some specific cause, which is probably a bacillus, a cell-growth rapidly forms in Peyer's glands, causing them to swell greatly until they present large, round, oval masses, projecting into the intestine, and at the same time amongst them smaller and rounder masses of the size of peas form in the solitary glands. The mesenteric glands are at the same time enlarged, congested, and softened. It is about the tenth or twelfth day that we see these glands in their highest stage. The microscope shows the new material which is deposited in them to be composed of round-cells or leucocytes. It was thought at one time that these cells possessed a special character, but if so they present no distinctive features to the eye. It has been said of late that a specific bacillus may be found in typhoid, and the report from Cornil's laboratory is that it has been discovered also in the liver, lungs, and spleen. The difficulty in proving this has been to get a pure cultivation, and to separate the specific form from other bacilli.

On section their structure is soft and medullary looking, and microscopic examination shows that the swelling is due to hypertrophic enlargement of the follicles, which have the structure of lymphatic glands. The same change, however, invades the submucous tissue around and beneath, and we have seen the small cells infiltrating the muscular and subperitoneal layers. Still its chief and primary seat is in the follicles and the tissue between them; and hence the disease culminates towards the death of this follicular layer and its separation *en masse* as a slough from its bed. The detachment is in a plane of demarcation, which cuts the Peyer's patch off, generally from beneath first, and afterwards at its edges. Thus, at one stage it may be found attached only at its edges or only at a part of its edge, or during life it may be found in the fæcal discharges as a cast-off sequestrum. In a bottle we have such a patch, which was so found in the stools, and which corresponds to the vacant Peyer's patch discovered afterwards on the death of the patient. Often, however, the separation is piecemeal. It is thus rather a sloughing than an ulcerative process.

When dead and not detached such sloughs take up the yellow bile-pigment from the bowel contents, and look yellowish or brownish, producing a very characteristic appearance of the diseased patches, attached as they are around the "ulcers" laid bare by the partial separations already completed. In milder degrees of the affection the typhoid change may pass off without sloughing, the typhoid swelling being absorbed, as it always is in the mesenteric glands. These milder degrees are, as we have said, observed in the patches higher up the bowel. We might ask, Are these higher patches which thus appear less affected, really only later attacked, so that in time they would be as bad as the lower, or are they less severely affected from the beginning? However late the death may be in the fever, we always find the gravest disease at the lower end of the ileum. For instance, perforation in the fifth or sixth week occurs in a lowest patch, when the upper patches are then only slightly affected. At this time indeed we find all the patches completely free from typhoid sloughs, except in the rarer cases wherein the typhoid has relapsed, when we may find disease of evidently two dates. We have once met ulcers of three dates with a well-marked history of triple typhoid fever.

The peculiarities of the "typhoid ulcer," resulting from the separation of these sloughs, are quite sufficient to distinguish it from all other ulcers, even from tuberculous ulcerations, which also affect Peyer's patches. Thus :—The peculiar yellow slough will be found around and on the patches more or less loosely attached. The ulcer left by detachment of the slough has a very thin floor, in which we generally see transverse muscular fibres, whereas the floor of a tuberculous ulcer is always much thicker than the surrounding intestine, however deep the ulcer may look; it also has tubercles in the subperitoneal tissue outside. The affected parts hold to the figure and position of Peyer's patches; but tuberculous ulcers, when old, run transversely around the inside of the bowel. We must, however, note that in old cases of typhoid ulcer, as indeed in all long-standing ulceration of the bowel, the ulcer spreads transversely.

The very thin floor of a typhoid ulcer may slough or be perforated at one or several small holes, or by a large rent, so that general peritonitis is set up by extravasation of fæces, or the same result may rarely be reached by extension of the inflammation through the walls without sloughing or perforation. On the other hand, more than one patch may be perforated. In very rare cases the extravasated contents are encysted to form an abscess, which may then behave like other fæcal abscesses. The typhoid ulcer may open vessels, producing grave and perhaps fatal hæmorrhage.

Next we must notice that the process may be very severe in the large intestine, its solitary glands being affected like the solitary glands of the ileum, so that fatal perforation may occur there, especially in the cæcum. Even the cæcal appendix may suffer typhoid ulceration, as we have sometimes seen; and Dr. Murchison has recorded a case in which it was perforated; so, too, we have seen a typhoid ulcer occur within a cæcal diverticulum of the ileum. We have a remarkable specimen where the whole surface of the colon is affected as if by smallpox.

But besides peculiarities due to the *stage* of the disease, we must notice that sometimes the *degree* of affection in the bowel is remarkably slight, say with only half a dozen small spots affected characteristically. There are all degrees of severity, from this to a state wherein the interior of the lower twelve or eighteen inches of the ileum is converted into a purple and grey sloughing pulp, while the mesenteric glands are in the same condition. It is obvious therefore that the fatality of the fever does not vary as the enteritis. A further proof of this we find in the converse cases, where patients following their ordinary avocations suffer typhoid perforation of the bowel while carrying a load in the street, &c.

We have met with a distinct variety of typhoid patch where the growth formed bold fungous masses, of unusually firm texture and with little tendency to slough. We met with a very marked example of this form of typhoid affection in a man who had extreme syphilitic and lardaceous disease of the viscera.

The typhoid ulcers usually heal up very quickly. A month or two after the subsidence of fever there may be scarcely any discoverable trace of thickening or other deformity of the patches and solitary glands; and four months after no sign of the typhoid except a partial deficiency of the patches.

**Tubercular Disease and Ulceration of Intestine.**—This is, perhaps, the commonest form of disease to which the intestine is liable, and it is remarkable that the same parts are affected as in the fever of which we have just been speaking. The disease is most frequently met with in conjunction with phthisis, and it is then that it can best be studied, since all its phases are constantly present. In an early stage all we can find is, on examining the lower end of the ileum, small, round, tuberculous deposits in the mucous membrane, corresponding mostly with the solitary glands; in the Peyerian patches also similar small round deposits may be found; at a later period these deposits are seen softening in their centre, and soon a small round excavation is formed, the primary lenticular ulcer; in the floor of this excavation other tubercles form, and opening into it compose a secondary or compound ulcer. The peculiarity of this process is, that as the ulcer deepens its floor thickens by new formation, so that perforation very rarely occurs. Only three times have we seen tubercular ulcers perforate the bowel; one of these was in the cæcal appendix, which is not unfrequently affected. Tubercles, however, still form in this thick base of the ulcer, extending into the subperitoneal coat, where they very rarely indeed, however, set up tubercular peritonitis. They usually appear in the course of the lacteals in an arborescent form and nodulated, running over the intestine to the mesenteric glands, in which usually secondary tubercles are found. These ulcers, like all long-standing ulcerations in the bowel, run transversely around the canal, forming at last broad raw bands. Sometimes healing occurs, but reopening of the ulcer is then likely. Occasionally the puckering from the healing process may induce internal strangulation. This may result through the contraction of the ulcer in healing, and it may take place when it is not quite healed. One such ulcer had contracted adhesion to the back of the bladder, and a loop of intestine became strangulated under it, the case being mistaken during the earlier stages for cystitis. Sometimes the coats of the intestine around and between the ulcers undergo acute inflammation, like the lung around vomicæ in pneumonic phthisis. We have already mentioned the small follicular abscesses in the coats of the intestine; these, though not tuberculous, generally occur in phthisis. The commencement of the large intestine also generally contains similar ulcers, and a very favourite site for ulceration is the ileo-cæcal valve; also, if the appendix vermiformis be opened, some ulceration may be found within it. Sometimes we find tubercular ulceration of the colon, when the small intestine escapes. Characteristic bacilli may be found in the edges of the ulcers.

**Dysentery.**—This is generally regarded as a specific disease, and probably due to malaria, being so often associated with disturbance of the liver, spleen, and other organs. It is believed by some, as the late Dr. Baly, to commence by an affection of the solitary glands; this is the opinion of many of those who have seen the disease in tropical climates, although not the opinion of all. Some hold the simplest form of dysentery to be an inflammation of the whole mucous membrane, affecting especially the follicles, and leading to great swelling, vascularity, and secretion from the membrane, and subsequently ulceration, identical, indeed, with what we have already spoken of as simple colitis; but at other times dysentery appears in a croupous or diphtheritic form; and therefore in dysentery we may meet with a *simple inflammation* of the mucous membrane ending in ulceration, or a *diphtheritic inflammation*, or a disease beginning in the *solitary glands.* In the most rapid and fatal form there is no particularising such distinctions, but the mucous membrane becomes infiltrated with blood, and its tissue perishes either in great patches or in isolated spots. Thus, true epidemic dysentery includes all the inflammatory conditions of the large intestine, so that the diseases already described under the name of colitis are anatomically like dysentery, and we may look upon them as capable of passing into it under suitable epidemic conditions. In an acute form of the disease, then, we see the mucous membrane of a deep red colour, swollen and pulpy, or soft, so that it can be easily torn, when some exudation would be found beneath it; the surface is covered with purulent mucus, and the solitary glands appear as numbers of small ulcers regularly placed. The ulcers grow more distinct and their sides become elevated by thickening; they are found especially on the summits of the rugosities of the bowel. The infiltration then advances farther, the ulcers becoming deeper, and the inflammation perhaps extends through the coats of the intestine, which swell to a thickness of several lines, and at the same time are soft. At this stage the peritoneum may be reached and peritonitis be set up, or sloughing may occur with the same result. The severity of the disease depends upon its extent; beginning at the rectum it may involve the whole of the colon, and in some cases include also the ileum. Microscopic examination of the tissue shows all the coats, but very especially the submucous, more or less crowded with wandering lymphoid cells, while the proper elements are cloudy and granular. If the person survive, the disease afterwards becomes chronic, and then the more elevated parts of the rugosities show deep ulcerations, while the parts that were in the recesses between the rugæ remain less affected; and thus the disease presents the appearance usually seen when coming under notice as a chronic affection in sailors who have arrived from abroad. In fatal cases of this kind, we find the colon extensively disorganised; the interior presenting a most irregular surface, from erosion by the ulcers; these may be isolated, and scattered over the membrane, but more usually they are united, and form a continuous serpentine ulceration throughout the large bowel, reminding us by their distribution of the rugæ by the destruction of which they arose. The floor of these ulcers is the muscular coat, or, if any repair is going on, an adventitious hardened lymph which has formed on it. Between the ulcers the mucous membrane is raised, red, and soft, in the form of so many islands. These may become greatly swollen and thickened, while they are still covered with the follicular mucous layer at their summits; they then look like crowds of polypi, and the condition is called *colitis polyposa.* If the curative process be at all present, the edges are vascular and indurated, the wall of the intestine is, at the same time, thickened by adventitious fibrous structure in the submucous tissue, and sometimes by hypertrophy of the muscular coat; the latter, however, may only be apparently thickened, owing to the contraction of the gut. These ulcers may heal, and then we find the various stages of cicatrisation present, or sometimes the intestine altogether healed. In the healing process a lymph is exuded on the floor of the ulcer, which becomes a hard callus, and which, contracting, draws together its walls, and thus we may often find cicatricial patches where the tissue appears puckered and radiating to a centre; when quite closed, all that is left is an unequal surface of a slate colour, these healed ulcers generally presenting a darker hue than the healthy mucous membrane, due, probably, to the change which the hæmatin has undergone; most of the pigmented changes found in membranes after inflammatory processes have their source in the chemical changes in the colouring matter of the blood. When a whole suface has been ulcerated and healed, it presents a very irregular and puckered aspect, and the hue is more or less of a slate colour. On the cicatrix we can no longer find the beautiful areolar surface natural to the colon, which presents itself even to the naked eye as a minute stippling, and is very easily seen by a lens, the mucous membrane being in fact destroyed. To examine the state of the membrane, we place a piece of the gut in a plate under water, when we can observe better the integrity or not of the surface. We have seen some cases where the ulcer has penetrated the colon, and set up a fatal peritonitis. The occurrence of hepatic abscess with dysentery we shall mention presently. Old dysenteric ulcers may reawaken into activity.

*Anthrax.*—As in the stomach there may be found in cases of anthrax peculiar-looking nodules Some of these may be black and others sloughing. These mucous black sloughs are seen in fresh specimens to be surrounded by a considerable amount of translucent œdematous swelling.

*Lead poisoning*—Dr. Fagge showed in two cases of lead poisoning where the patients had suffered from colic, large slate-coloured patches on the mucous membrane of the large intestine. A similar condition may be found after the prolonged action of other minerals. There is a remarkable specimen added to the Hunterian Museum by Dr.

Williams, taken from a lady aged seventy-four who for forty-three years swallowed a grain of calomel every night. The mucous surface of the colon is black from the deposit of mercury in the submucous tissue.*

**Syphilitic Ulceration.**—In women the subjects of syphilis the rectum is sometimes found ulcerated at the lowest part for a space of several inches, the ulcer, perhaps, running up into the sigmoid flexure. Such ulceration appears to depend on extension of inflammation along the perinæum in the primary stage of syphilis. The interior of the bowel is much destroyed; and fistulæ may result. The ulceration may pass in and out behind the mucous membrane and even deeper, so as to convert the wall of the bowel to very much the appearance of the apex of the right ventricle through the number of fleshy columns that cross it. A stricture of the rectum may result, and sometimes the bowel wall is perforated and retroperitoneal abscess set up. Such ulceration of the rectum is not often seen in males. Primary syphilitic sores are occasionally met with here.

**Morbid Growths.**—*Cancer.*—Secondary cancers ef epithelial, carcinomatous, or sarcomatous kinds appear in the small as well as the large intestine, as little rounded patches seated in the mucous membrane; they are not unfrequent, but are apt to escape notice. We have known this form of cancer of the colon to be secondary to cancer of the liver, reversing the usual order of occurrence, in which the liver is infected from the colon. One also finds the intestine invaded and opened by cancer in neighbouring organs, thus the ileum, or, much more often, the rectum by uterine cancer; the colon by cancer of the stomach, gall-bladder, kidney, &c.

As to primary growths, the remarks we made on the varying degrees of malignancy, and therewith of softness of cancerous growths in the stomach, apply equally to such growths in the intestine.

*Lymphoma or adenoid carcinoma.*—We must note that the small intestine is very little subject to primary cancer, and when this does occur it nearly always has the form of lymphoma; that is, it has a structure almost identical with that of lymphatic gland. It always then affects the mesenteric glands, enlarging them enormously, the tumour extending also to the lumbar glands; columns of the growth in the course of the lacteals generally connect the cancer in the bowel with the enlarged glands. This kind of cancer has three chief peculiarities:—First, it rarely ulcerates; second, it rather widens than narrows the channel of the bowel; and third, it shows very little disposition to undergo degenerative changes. To the naked eye it appears as a medullary cancer; its growth is rapid. Lymphoma may also affect the colon, but less frequently and less largely.

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* 'Path. Trans.,' vol. xviii.

Lymphona of the submucous coat is also found in association with leukæmia; it then has the same microscopic character as when the blood is unaffected, but it is usually in smaller patches and the glands less implicated. In one case of Hodgkin's disease large flat tumours grew from the inside of the intestine.

In the *colon* primary cancer is frequent; it affects chiefly the cæcum or sigmoid flexure. One of the most interesting forms is *colloid*, which has here the same characters as in the stomach. The gelatinous matter appears in some cases to be formed as an active outgrowth of the follicles of the mucous membrane. Colloid forms a thick mass of cancer, generally extending all around the bowel for several inches of its length; it may spread over and distend the peritoneum. On the other hand, sometimes the bowel is attacked by colloid which was primary in the peritoneum. It has less tendency to ulcerate and contract the bowel than carcinoma, but more than lymphoma.

*Cylindrical epithelial cancer* is relatively frequent in the large intestine, and may be combined with colloid; its structure is often a beautifully perfect follicular tissue, like that of Lieberkuhn's follicles, and its surface may grow into papillary processes; it may set up secondary cancer in the liver, which shall have exactly the same structure, that of intestinal mucous membrane. This disease in the colon is generally circumscribed at a small spot, and it constricts the bowel there. It is most frequent in the sigmoid flexure and rectum.

In the latter region these new growths have been especially studied by Cripps. Whether styled scirrhous, medullary, or sarcoma, he finds a structure within similar to that of Lieberkuhn's follicles, and, therefore, such terms as malignant, semi-malignant, and simple adenoid might be a good division for them both histologically and clinically. The malignant involve the coats of the intestine, whilst the innocent project internally. The latter show an hypertrophied mucous membrane with a glandular growth beneath, the spaces lined by columnar epithelium with a basis of simple fibre or cells or spindle-shaped sarcomatous tissue. In simple mucous polypus there is an arborescent fibrous network on which are arranged epithelial cells.

The more ordinary forms of *carcinoma* also occur in the intestines. Lymphoma, which we have already mentioned, would usually be called "medullary cancer;" but we meet also with true carcinoma in its medullary form as well as in its scirrhous form, and it is to these as well as to the colloid and epithelial forms that cancerous stricture is usually due, while, as we just now said, the lymphomatous variety of cancer widens the bowel. In one case the intestine ran right through the tumour and was thickened and dilated. Carcinoma of the bowel has the same characters as carcinoma of the stomach. In the intestine the fatal stricture need not be an entire closure. Often we are able to easily pass the blade of the enterotome through a stricture which has caused fatal obstruction. No doubt enemata are sometimes sent upwards past a stricture, with the effect of increasing the tension above it. In one case there could be no question that five pints of fluid passed up through a stricture which yet continued obstinate and proved fatal. The mucous surface of this form of cancer is constantly found as an ill-conditioned sore with thick raised edges. The ulceration may lead to perforation, with the usual consequences of this, one of these being abdominal abscess. This is not uncommonly met with in connection with cancer, but it is not quite certain that the cancerous process ever passes into the suppurative one. It seems rather that the abscess is produced by the irritation of fæcal matter which has escaped from the diseased or perforated bowel.

We sometimes find cancer at more than one point in the alimentary canal, as the duodenum and rectum. We have several times found the stomach and the colon separately affected in the same case.

Sometimes we may meet with *sarcoma*, especially in the form of *melanosarcoma*, secondary to melanosis of the eye or skin.

Tumours of the muscular tissue, or *myomata*, may occur as in the stomach and project inwards.

We meet occasionally with *villous cancer* in the intestine; it has the same characters as in the stomach. Also patches of superficial villous growth of simple fibrous structure with vascular loops; these have no malignant character; they are called *papillary fibroma.* Such patches may arise in dysenteric ulcers.

Occasionally *lipoma* occurs in the submucous tissue of the bowel. It is generally in the form of little masses, which may project in a polypoid shape.

*Cysts* with mucous contents are found in the wall of the intestine; they are very rare, but they sometimes reach a large size.

We have already spoken of the occurrence of tubercular ulceration. This is the only form in which tubercle attacks the intestines, for they generally escape or are very little affected in general miliary tuberculosis.

We occasionally meet with growths of a *polypoid* form hanging from the mucous membrane. Their seat is generally the rectum, where they come under the notice of the surgeon. They are small round bodies, consisting of a fibro-cellular structure, and highly vascular. Sometimes they are composed of glandular tissue, like that of the mucous membrane, as we have already mentioned. Occasionally we meet with small polypoid growths in other parts of the canal, as we may see in our specimens. In one there are a number attached, forming a bunch of pedunculated tumours, very similar to those sometimes met with in the bladder. The solitary glands in the colon may enlarge and become pendulous.

*Hæmorrhoids* form a kind of tumour at and within the anal aperture. Their history is very simple. After death they generally look mild and innocent in comparison with the annoyance they have created during life; they go down with the disappearance of the vital turgescence. Hæmorrhoids consist essentially of varicose swelling in the submucous tissue of the rectum and anus. The swollen veins there are in connection with the proper venous system of the part, which we know is in free communication with the portal along the gut, and with the iliac system in the perinæum. The venous flow here has peculiar difficulties through gravitation, and through the pressure of fæces and of the internal genitalia, through irritation, &c., nay, even through muscular efforts of all kinds, for one cannot use his arms or even cough without bearing down upon the levator and sphincter ani, so as to check the freedom of venous current through those muscles. Hæmorrhoidal tumours are either all around the anus or limited to a part of it. They are either inside or outside, or partly in and partly out, and important surgical classifications for their treatment are based on their differences of site. But their pathological nature is always the same. They consist of dilated veins surrounded and separated by a lax fibrous tissue, and covered by mucous membrane or skin. It was held by some that the blood-cysts are cut off from the venous system; but this is not believed now, as under favorable circumstances injection from the veins can be effected. Sometimes the venules in them are numerous and tolerably even in size, at other times there is one great preponderating blood-cyst. Sometimes small arteries may be traced into them. Like other imperfections of the circulation, they are most apt to appear in adults as they are growing old; but sometimes they occur in the young, and they are sometimes hereditary or even congenital. Their chief importance arises from the accidents they are subject to. Thus, they are liable to be inflamed, and cause secondary trouble by sloughing and suppuration. We have already alluded to piles at the upper extremity of the alimentary canal in the œsophagus.

**Morbid Contents.**—*Concretions.*—These may have been secreted in the intestinal canal, and are then called *enteroliths;* or they may have been formed from matters taken into the bowel, and then are named *accidental concretions.*

We have already mentioned that calculi may arise in the vermiform appendix; the earthy matter consisting mostly of phosphate and carbonate of lime, the nucleus being fæcal matter or some accidental substance. In other parts of the intestine very large calculi may arise, as seen in our Museum, and sometimes cause considerable obstruction; in one case the concretion made its way through from the cæcum. In the lower animals very large stones may form, as those from the intestine of a horse; they often have a nucleus of oats, or consist wholly of vegetable matters. In one, composed of triple phosphate, the nucleus is a piece of iron. Some of the most remarkable of these are the bezoars, found in the intestines of the wild goat inhabiting the Persian province of Khorassan; these are soft, and contain a peculiar principle, called *ellagic acid.* *Accidental concretions* are those which form directly from substances taken as food or medicine, and thus masses of chalk or magnesia have occasionally collected in the inside; and we can show remarkable hair-balls, like masses of felt, taken from the stomachs of cows, and a somewhat similar mass of hair we have seen removed from the stomach of a lunatic.

We may also meet with substances which have not been formed in the canal, but have passed into it, as gall-stones—these sometimes being so large as to cause fatal obstruction; several substances which did so may be seen on our shelves, as coins, knives, stones, &c.

*Fæcal matters.*—These are especially worthy of notice when discharged during life, but also on the post-mortem table we should observe their *form*, *consistence*, and *colour*. It is not altogether correct to say that solid fæcal matter is found only in the large intestine, for it may be seen of some consistence at the end of the small intestine. The fact of *scybala* being present, as well as the colour or fluidity of the fæcal matter, should be noticed as sometimes of importance: if of a dark colour, this may be due to blood, or to medicine taken, as iron; but sometimes a chemical test or the microscope is required to decide its origin. We have sometimes met with large quantities of hardened fæcal matter; thus, a round lump, weighing 17 oz., was in the sigmoid colon in one case; there was slight peritonitis over the colon at its seat. If blood has been poured out from the stomach it is of a black colour, but if it came from the lower part of the bowels it is of its natural bright hue, or of a brownish colour. The blood may have come from above the stomach; we have met with more than one case of fracture of the base of the skull with laceration of both carotids where the blood extended throughout the whole alimentary canal and death was caused by hæmorrhage. The presence or absence of bile should also now be observed, as well as during life.

*Entozoa, or intestinal worms.*—The four commonest kinds are the *tænia*, or tapeworms; *Ascaris lumbricoides*, or round-worm; *Oxyuris vermicularis*, or thread-worm; and the *Trichocephalus dispar*. The most interesting part of the history of intestinal worms is their mode of development, discovered by von Siebold, Küchenmeister, Leuckart, and others; including in this country especially Dr. Cobbold. These observers have shown that the vesicular worms, known as hydatids, are but the immature forms of tapeworms. The sexually complete animal, or tapeworm, inhabits the intestine, while the incomplete animal, in the form of an hydatid, is developed in a solid or parenchymatous organ. Thus, instead of the ova of the tapeworm producing young animals of the same form, an intermediate stage of development occurs, in which the head of the creature grows into its proper shape, and has a short neck, but the end of this develops into a large cyst, into which cyst the head and neck can retract themselves. The head and neck are known as the "*scolex*." Thus arises the cystic variety of worm, so that it is an incomplete form of the tapeworm. Experiments on the lower animals show that if a cysticercus be taken into the stomach it will soon be developed into a tænia; and again, that the ova which are produced from this animal, if taken into the intestine of another, are not formed into tænia there, but are taken up by the blood-vessels and deposited in the solid organs, as the liver, heart, brain, &c., where the first stage of development into a cyst occurs. Should they then be eaten with their host by another animal, the perfect worm is produced within the intestines of the devourer. It has thus been shown experimentally that the *Cysticercus fasciolaris*, which inhabits the liver of the rat (a specimen of which we possess), if eaten by a cat, develops within her into a tapeworm. And again, that the ova of this tapeworm, if eaten by another rat, become cystic worms in its liver. In the same way the ova of the *Tænia cœnurus*, which inhabits the intestine of the dog, passing out with the fæcal matter on to grass, may be eaten by sheep; the embryo is taken through the circulation into the brain of this animal, and there becomes the *Cœnurus cerebralis;* this, if eaten by a dog, becomes again the *Tænia cœnurus*. Experiments on the human subject have also shown that cysticerci may become developed into tænia; and it has been proved that it is the cysticercus of the pig which produces *Tænia solium;* while *Tænia mediocanellata* is produced from the cysticercus of the calf and ox.

The echinococcus is a cystic worm, which develops in the intestines of the dog or wolf into a small tapeworm with very few joints, but this tapeworm has, we believe, never been found in man. Although the difference in form between a round cyst and a long worm may appear great, yet such a specimen as the cysticerus of the rat will show how one may pass into the other; for we see it has a long neck, and so has already somewhat the appearance of a tapeworm; and then, as regards the form of the head, we know that in both there is a circlet of hooklets which enables the creature to fix itself to the neighbouring structure for the purpose of nutriment. The *Cysticercus cellulosæ*, which is found in various parts of the human body, consists of an oval bag or cyst of thin membrane, containing within it a clear fluid, and from this cyst there protrudes a neck, which is of different lengths in different instances, and at the end of this is the head, which, like that of the tapeworm, has four suckers, and a circlet of hooks in the midst of them; the latter average twenty in number.

Tapeworms sometimes measure several feet in length. They are composed of a number of segments, each of these containing hermaphrodite sexual organs, and discharging innumerable ova. Although these are so far independent, they are all united, and gain their nourishment through the head, which is fixed to the intestinal wall of the host; from the other extreme the ripe segments drop off, like the fruit from a tree, whose root still remains in the form of the four suckers, fixed to the intestine. The head which is attached is a little knob at the end of a long flat filament. This differs in different species; there are always in *tæniæ* four suckers, arranged equidistant around the raised centre of the free extremity. In *Tænia mediocanellata* the suckers are black, and there are no hooklets, but in *T. solium* the suckers are pale, and there is in the centre between them a double circlet of hooklets concentrically placed, and circumscribing a small circle. In the former the joints are often depressed. Other varieties of tænia—*T. marginata*, *T. elliptica*—have been known to occur, but very rarely. The tapeworm of the Swiss, *Bothriocephalus latus*, has a smooth oval end, with a groove on each side, but neither suckers nor hooklets. It is asserted by Knoch that this worm does not require an intermediate host in its development, but there are great doubts of the accuracy of this view. There are numerous specimens of tapeworm on the shelves. Students often inquire, when patients bring portions of worms, whether these are from near the head or the opposite extremity. The head and neck consist of one long and very slender piece, and the segments which come next appear as very narrow pieces, then they increase in breadth until the middle of the worm is approached, when the breadth somewhat exceeds the length; towards the tail they become narrower and longer, each segment acquiring somewhat of a conical form, tapering to that extremity which is attached to the broad end of the segment before it. The place of growth of the ordinary tapeworm is the small intestine; we may find one after death occupying several feet of the intestine, stretched out at full length, the head directed upwards, but free and moving about, as if seeking for some new place of attachment.

*Trichocephalus dispar.*—These are small hair-like worms, one or two inches long, the pointed end constituting the head, and they lie curled up as we see in our specimens. They are met with mostly after death in examining the intestines carefully, and it is said that in France nearly every body contains some in the cæcum. Although we have occasionally seen them, we cannot say that this is the case in England; indeed, they are somewhat rare here. We have a drawing of one which we examined not long ago; it is a male specimen, and shows at the blunt end the sexual organ projecting from a sheath. These worms are not known to produce any symptoms.

*Ascaris lumbricoides* is the common round-worm resembling the earth-worm, and often called lumbricus; it is developed in the small intestine, where we not unfrequently meet with it in considerable numbers, especially in children; three hundred to five hundred are said to have been passed by one person. After death (as during life) the worm may pass into any part of the intestinal canal, as the stomach or œsophagus, or it may pass out from the mouth or nose. Cases are recorded where one got into the larynx and caused choking; also where the worm penetrated the intestine, causing fatal peritonitis, or pierced its way into the abdominal wall, and there formed an abscess, or was found in the pleural cavity, or sometimes one of these worms has passed up the bile-duct.

*Oxyuris vermicularis*, the well-known thread-worm, is developed in the rectum, sometimes in enormous numbers.

*Ascaris mystax* of the cat has been shown by Dr. Cobbold to be occasionally found as a parasite in man.

For the anatomy of this and the other worms we must refer to zoological works, especially the excellent monograph of Dr. Cobbold. It appears that the ova of round-worms pass out of the body, and are very slowly developed when they reach water; but it is yet uncertain how they obtain access to the human body, and especially whether an intermediate host is necessary.

*Bilharzia hæmatobia*, a parasite residing in the veins, though it more especially affects the urinary system, is found also in the intestines, where it produces extravasation of blood with thickening, ulceration and fungoid projections arising in the mucous and submucous tissues. It is endemic in Egypt, and to a less extent at the Cape of Good Hope, producing a very terrible form of disease.

A minute fluke-worm, less than a line long, *Distoma heterophytes*, was found in hundreds by Dr. Bilharz, of Cairo, in the intestines of a boy.

*Sclerostoma*, or *Anchylostomum duodenale*, occurs in South America and hot climates, and is supposed to give rise to the Egyptian anæmia. It caused a fatal illness in many of the workmen engaged on the St. Gothard Tunnel (see Weber, 'Path. Trans.,' 1867).

# DISEASES OF THE LIVER

**Malformation.**—Sometimes the liver has an unusual shape, so that the whole organ is rounded or the relative size of the lobes is unnatural. It is often very doubtful whether such changes are not rather the result of bygone disease than of original malformation. We know from actual experience that the drying up of an abscess, or syphilitic deposit, &c., will very much alter the form of the liver in adult life; but we can infer that a much larger extent of change will arise from the like disease at earlier periods before growth is complete, and thus we should think that in a liver which consists almost entirely of a large left lobe, the morbid condition that caused the atrophy of the other part must have been in action at a very early period of existence.

Sometimes an additional lobe is present in an otherwise quite healthy liver; we have seen it occasionally on the under surface of the right lobe. Projecting lobes are present very constantly in the livers of some kinds of animals, such as rodents, and yet the shape of the liver would appear to have no importance which would correspond to such constancy of its figure in any class of animals. Certainly the form of the liver is very commonly surprisingly altered by external influences, especially by tight lacing. When this is extreme, the right lobe may have its upper surface thrown into folds by the circumferential pressure, the convex surface yielding more easily than other parts. But the usual effect of tight lacing on the form of the liver is this—that a deep transverse furrow runs from side to side across the lower surface of the right lobe; it is deepest at the right border, where it may divide the mass of the liver more or less definitely into two parts, one in front of the other. The liver has naturally a somewhat bent figure, which is arched from before backwards, the arch fitting into the diaphragm. Circular pressure around the arch increases its convexity, bending it up in the middle, and compressing the middle part in the concavity of the arch, so that the substance here atrophies and wastes away, thus producing the transverse groove described. This groove may run across to the left lobe, and may be so deep as to completely divide the anterior from the posterior part, fibrous remains with vessels, &c., alone connecting the two portions. Thus, what appears to be a movable tumour in the region of the liver may be produced, for the organ is generally thrust down in these cases, so leading to serious errors in diagnosis. The groove itself is always more or less fibrous looking, in consequence of loss of the secreting tissue.

Men who wear straps round their waists suffer a degree of the same change, but in men the effects of this lesser pressure usually are different. A groove or depression with a fibrous appearance runs along above the anterior edge of the liver in front, where the false ribs press upon it, the whole of the thin front edge being also more fibrous than natural. The contrast between the anterior and posterior part in these cases in point of consistence is very remarkable in elderly persons. This sensitiveness of the liver to pressure clearly shows the necessity for its protection by the sheltered position which it naturally occupies. It is not uncommon to see on the left lobe a round patch of superficial fibrous wasting adjacent to the heart, and caused by the constant pounding of the liver by the heart against a flatulent or overfull stomach.

White marks on the liver, which often attract the attention of students, are due to accidental pressure after death and can be made with the fingers. Some German writers speak of the "floating" liver, or a liver very movable on account of relaxation of the ligaments.

**Hypertrophy.**—The liver is often found to be large, and yet apparently healthy in texture. Analogy would lead us to expect such a result to arise from over-use of its function, We cannot say to what size the liver may reach from such a true hypertrophy : we have met with as much as 19 oz. of healthy kidney structure in persons who have drunk enormously of beer, and in similar examples have found the liver to reach 80 oz. weight, while its texture appeared healthy.

The term hypertrophy was formerly very loosely employed for any uniform enlargement of the liver ; but we now recognise enlargements from engorgement, lardaceous disease, or other change, in the light of our knowledge of such diseases. The swelling which these produce is not an hypertrophy of liver tissue, but an introduction of some other matter into it. Simple hypertrophy is practically of little account ; it is most certainly witnessed in cases where parts of the liver have wasted—for instance, in the tight-laced liver just described, where the groove is deepened by thickening of the rest of the organ ; or when syphilitic or other local destruction has caused shrinking away of large parts, say not unfrequently of the entire left, or even of the right lobe, the remainder then will become enormously enlarged. The lobules are coarse ; they appear to increase, and not to multiply. We have seen the whole right lobe removed, and the left then weighing 56 oz.

Occasionally we meet with outgrowths apparently composed of hepatic tissue. If this be so a real proliferation of the true liver structure is possible. These, however, are usually styled "adenoma," under which head we shall refer to them again. Of late the term *hypertrophy* has been applied to the case where the hepatic cells increase in size, and *hyperplasia* where the hepatic cells increase in number.

**Atrophy.**—The diseases that are characterised by extreme wasting of the hepatic tissue are so important that we must give careful attention to them. Many of the diseases which we shall subsequently consider include more or less atrophy among their results ; and in some, as in cirrhosis and the ramose atrophy of nutmeg liver, this atrophy is an important part of all the change. Of this wasting we shall, however, speak in describing those kinds of disease.

The liver may be found wasted in cases such as cancer of the œsophagus, where the cause of general wasting is of a simply starving kind. But there are other diseases, such as phthisis, in which, however wasted the whole body may be, the liver is commonly large ; and this because the organ, such as the lung in phthisis, whose disorder leads to the wasting, gets help from the liver when its function is impaired, thus causing to the liver over-work and hypertrophy. In cases of capsulitis where the liver is enveloped in a thick white covering the organ may be found shrunken to half its natural size.

The diseases in which a simple atrophy of the liver forms the main characteristic feature are three, and are named, after the colour the liver shows, respectively—yellow, red, and brown.

*Yellow atrophy and red atrophy.*—The disease which includes these conditions is well defined, and yet anomalous, both clinically and anatomically. For though probably inflammatory and nearly always connected with unmistakable sub-inflammation, it nevertheless neither arises out of nor runs into the common forms of inflammation, viz. the ordinary chronic inflammation of cirrhosis and the intense inflammation of suppurative hepatitis ; further, the capsule of the liver in acute yellow atrophy, as a general rule, is not inflamed, though acute inflammation of most organs commonly involves their capsule. Still, in occasional instances inflammation of the capsule is present with general peritonitis ; the bile-ducts and gall-bladder, also, while they vary a good deal in their state, often show a remarkable redness within, suggestive of inflammation, and they contain sometimes peculiar tenacious mucus.

Considering all these circumstances, we cannot set down yellow atrophy as unqualified acute inflammation, and yet, as we shall see, sub-inflammatory change around the portal vein causing red atrophy is so commonly, if not as we believe, constantly met with in it, that the connection of yellow atrophy through red atrophy with inflammation must be held as undoubtedly made out.

The liver with yellow atrophy is small, often very small, weighing less than one half or even only one third of its normal weight. The organ is diminished equally in all directions, so as to preserve its natural form ; but in consistence it is remarkably soft, supple, and flabby, the capsule wrinkling loosely from want of the natural bulk of its contents, and the organ when placed upon its edge doubling down upon itself, unable to sustain its own weight. This doubling down may occur in the living body, the front of the liver falling back from the abdominal wall, while the colon rolls up into its place, causing entirely to disappear the usual anterior area of hepatic dulness.

Some of these livers, however, have shown partial and unequal distributions of the yellow atrophy, intermediate parts being in the state of red atrophy, of which we shall presently speak. Under these circumstances the liver may be marked by uneven elevations, from the shrinking of the red atrophied parts out of proportion with the yellow. The outside is generally of a dirty greenish-clayey colour; but on section the colour is bright yellow, like moistened rhubarb, and in all the cases we have seen there has also been present some red atrophy in the section; this is found around the great vessels, especially the portal veins. The outlines of the lobules on section can scarcely be seen, either in the yellow or red part; but if seen at all are seen in the yellow part better than in the red, and the lobules are then seen to be much larger in the yellow than in the red. In those cases which have lasted longest the red portion is in greatest quantity; and this fact, taken with the microscopic characters of the two portions, shows that the red is a late stage, or chronic form, of the yellow atrophy, the yellow being an acute or excessive intensity of the red. A greyish material may also be seen between the lobules in the tracks of the portal venules; this is an intermediate stage between the red and yellow stages, and signifies an incipient inflammatory thickening, of which the red condition is the end. There is nothing remarkable in the appearance of the channels of the blood-vessels, but it is said that injection will not run through the capillaries, breaking rather into the parenchyma of the lobules.

The microscopic appearance of the yellow atrophy is that which is best known; it reveals a destruction of the hepatic cells which are found as granular relics in the lax and flocculent remains of the capillary network, little of the tissue structure remaining to be seen. But it is important to note that the yellow portion always shows some tolerably healthy lobules, and parts of lobules, whose cells persist, but are coloured bright yellow with bile-pigment. The process thus revealed is one of simple destruction of the secreting elements of the liver without any formative changes, this evidently being a rapid proceeding, corresponding, indeed, with the fearfully quick course of the grave symptoms in acute atrophy.

On the other hand, the red atrophy shows a very different appearance. The vascular network of the lobule is thickened much and shrunken, at the expense of the spaces which should contain the liver-cells. In these spaces small nucleated cells are found in parts, like epithelium; these appear to be degraded liver-cells, which have lost their proper character without perishing entirely; but for the most part the spaces contain only a few granular relics. We have seen in the thickened substance about the capillary network a quantity of pus-like cells, crowded in it around the portal capillaries. Thus, in the red part an inflammatory condition is proved to exist, not only by these pus-like cells in certain cases, but also by the thickening of the vascular stroma which resembles the thickening of it in cirrhosis; differing from this chiefly in two points, first, that it extends more uniformly throughout the lobules instead of being limited to the tracks of the portal vein, as in cirrhosis; and secondly, in the destruction of the liver-cells being greatly beyond what the pressure of this thickening will explain.

Some have even described a formation of new ducts, but this may have been due to a false appearance and to the shrinking of hepatic cells. The pathology of the disease is therefore somewhat obscure, for, whilst there is an evident rapid destruction of the hepatic tissue, there appears to be a tendency to new formation both of cells, nuclei, and connective tissue.

Besides these changes in the structural constitution of the liver its chemical constitution is also changed. It may contain some fat, but generally this is present to no important extent. The peculiar character is in the presence of large quantities of leucin and tyrosin. The proper crystalline forms of these substances may be seen in the tissue, and may be found in the blood of the portal or hepatic vein. They may be absent in fresh specimens, and yet be found in large quantity when these specimens have been kept, with or without preservation in spirit. In warm weather we have seen a white efflorescence of these substances form on sections which have been for several hours exposed to the air.

The course of this disease is usually swift, lasting from three to five days, and characterised by a combination of grave cerebral symptoms with jaundice, hæmorrhages, and moderate fever. About two thirds of the patients are females, and of these many are pregnant. Intense emotional disturbances have appeared to cause it, and it has occurred during the fever of syphilis.

In various chronic affections of the liver an acute fatal atrophy may supervene. In many instances after rapid illness, besides the evidence of acute atrophy, a long-standing cirrhosis has been found. In one case of jaundice, arising from gall-stone and accompanied by an enlargement of the liver, there was a rapid subsidence of the symptoms with shrinking of the liver, and an onset of fever, causing death in four days. The liver was found to have undergone atrophic changes, and the gall-bladder was full of calculi. Klebs has found bacilli in the bile-ducts, but Cavafy, in a communication to the Pathological Society, has failed to confirm this statement, so also Dreschfeld. Dr. Goodhart has seen the disease in a child aged two and a half years, and Dr. Samuel West also.

Besides the change in the liver, the heart and other muscles generally have their fibres in a state of fatty degeneration. The kidney also is commonly loaded with fat and deeply jaundiced, while crystals of tyrosin and leucin are present in it. Such a general distribution of fatty degeneration in the muscles, kidney, &c., is sometimes termed *steatosis.* The urine contains leucin and tyrosin. Later observers have found also minute changes in the brain, and so they say an explanation is afforded of the cerebral symptoms which had before been put down to blood-poisoning through the liver or kidney.

*Phosphorus poisoning.*—In these cases some German observers have described the liver as corresponding with that of acute atrophy, and indeed, one authority has gone so far as to suggest that all cases of acute atrophy may have had phosphorus poisoning for a cause. In this country the few cases of poisoning which have been observed have not borne out this view. The liver, unlike that of atrophy, has been of full size, firm, and feeling like a fatty liver. The microscope has shown that a fatty change has taken place to a great extent, but the secreting cells have still remained intact. In some cases they have been found cloudy or granular, and the whole arrangement of the secreting cells has been disturbed. On the Continent, where more cases have been observed, it is said that the epithelial cells of kidney, stomach, and other parts show a fatty degeneration.

*Brown atrophy.*—By this we mean the disease which has been called chronic atrophy by Frerichs, and red atrophy by Rokitansky, both speaking of it as dark brown in colour. It is an entirely different kind of disease from the yellow and red atrophy we have just described, its symptoms being those of obstructed hepatic circulation without jaundice ; symptoms, in fact, identical with those of cirrhosis, but often accompanied with severe diarrhœa. The liver is small ; we have found it weighing only 28 oz., but it has been found only 24 oz. in weight. The organ is simply diminished in size, with perhaps its edge wasted and white looking. It is flabby, soft, but not so pulpy as in the yellow atrophy. There is no appearance of increase of fibrous tissue within or without, the capsule being thin and the substance dark brownish red—that is, of a deep liver colour. Microscopic examination shows the cells of the liver exceedingly small and charged with brown grains, many of which are set free by the bursting of the cells, the nuclei of which are generally plainly visible. Around the portal veins the microscope shows some thickening of Glisson's capsule, and we have seen on close observation of the section little grey, tubercle-like points, microscopically composed of remains of liver tissue, in which all the veins were occluded. Attempts to inject the liver from the portal vein under these circumstances more or less entirely fail. There is a deficiency of bile. Brown atrophy has been known to occur in early life, but it generally appears about or after middle age, and it has been found associated with pigmented spleen from intermittent fever.

**Congestion.**—Being a highly vascular organ,—microscopic sections of injected liver look as if there were nothing but blood-vessels in the liver,—the liver is subject to great congestion, which during life may enlarge it perceptibly, as we often observe in heart, or lung, or any ischæmic disease. At a post-mortem examination, however, a fulness of the liver with blood—that is, a simple congestion, proves nothing whatever.

No doubt there is such congestion of the liver as is popularly understood, that is, a bloated state of the organ, produced either mechanically, by sedentary habits, or by more direct sources of obstruction; or passively, by torpid vein stream, suppressed discharges, &c.; or actively, by irritating foods, hot climates, miasms, fevers, and so forth. All this may be, we say; and so, no doubt, congestion is a useful medium of recognition for such cases during life. But after death such moderate congestion reveals simply nothing whatever, and we must remember, in thinking of these states, that they are very various and often complex, and the mere effect upon the blood-flow is a very minor consideration, although convenient in giving a common and well-understood name to the states which cause it.

The only congestions which we can recognise after death are the more intense *mechanical* congestions, and to these we wish to draw attention. They arise from obstacles to the onflow of blood in the hepatic vein, producing an obstruction whose effect is to widen the main trunks of those veins and then their branches, until the capillaries of the hepatic veins, which are in the centres of the lobules, become distended. These capillaries converge from the circumference of the lobules, where they arise from the portal venules, gathering themselves together upon the intralobular hepatic venule which runs down the centre of the lobule.

Now, when this distension of the capillaries of the hepatic venules about the centres of the lobules has been only moderately severe and prolonged, they become dilated, and are not able to empty themselves even when the blood has got out of the great vessels in the removal of the liver from the body. Under these circumstances the section of the liver shows more or less dark centres to all the lobules, while the circumference of the lobules are pale, for the capillaries near the portal vein are not thus distended, but anæmic; hence there arises a minute mottling, with dark and light parts more or less evident, and this state is common in death by chronic bronchitis or other diseases, wherein a varying and inconstant obstruction to blood-flow has existed.

Dr. Wooldridge has shown that when certain proteid bodies are injected into the blood infarction and thrombosis of the portal vein is produced; also if the liver be injected with fibrinogen a fatty degeneration takes place.

*Nutmeg liver.*—But when this distension of the hepatic capillaries has been long in great force, as in fatal mitral obstruction, then the above-mentioned darkening of the centres of the lobules, contrasted with paleness of their circumference, becomes wonderfully strongly marked; the light and dark parts being sharply defined, so that the mixture of colours, without mingling, gives exactly the appearance of the cut surface of a nutmeg, the intricately folded and twisted lobulation revealed in the liver corresponding curiously with the similar twists and folds in a cut nutmeg.

The great and permanent distension of the hepatic capillaries, which are close set and radiating, presses severely on the columns of hepatic cells which occupy the radiating spaces between the radiating vessels, and this pressure destroys gradually the hepatic cells, from the centres of the lobules outwards; while these capillaries widen much, and, containing dark blood, make a very black area on section. Meantime, whatever be the reason of it, the bile-pigment in the more central hepatic cells of the lobules is always in much greater quantity than at the circumference, perhaps because the distance which the bile has to travel to the bile-ducts on the surface of the lobule is greater from these centrally placed cells. Thus much for the dark central areas coloured by dark blood and bile-pigment. The white part, on the other hand, is always on the circumference of the lobule, and in it with care we are sure to find the little line indicating the portal venule with its so-called Glisson's capsule around it. The portal capillaries, like arterial capillaries, to which they correspond, are empty of blood, whence the whiteness partly arises; but the whiteness is also due in part to the presence of a quantity of fat in the liver-cells about the portal vein. It appears that the liver-cells have such an attraction for fat, that those nearest the supplying vessel take it all up and allow little to pass on to the deeper cells, over whom they have the advantage of position. Thus, the whiteness about the portal venules is due to anæmia and fat. A yellow tint is always present from engorgement of the bile-ducts, which we know goes so far as sometimes to cause jaundice in heart disease. In both the white and dark parts, but chiefly in the dark, the microscope often reveals blood-crystals lodged in the tissue, from bygone extravasations. And not unfrequently blood-black patches in the dependent part of the organ show more recent extravasations of blood, which may reach some size, and be called *apoplexy of the liver.*

Some authors have described, as a result of long-standing congestion, a proliferation of cells and production of connective tissue, which, continuing to increase, at last produces a true cirrhosis. For example, it is said that in mitral disease such a change may be wrought in the liver as to constitute at length a substantive cirrhotic state, with its accompanying ascites. Cornil and Ranvier support this opinion, and style the affection *cardiac cirrhosis.* The expression is constantly met with in French journals. Such a result as this we have never seen.

*Ramose atrophy.*—If heart disease has been at once unusually long and severe, the effect on the liver may go yet further, and the tissue of the centres of the lobules may be reduced to a state like that of cavernous tissue, nothing but dilated blood-vessels remaining. We then find that the smaller hepatic veins surrounded by these dilated capillaries come to view on the section as deep red ramose lines, sunken on the section on account of the wasting around, and in many parts, the process of distension and destruction having extended quite through some lobules and segments of lobules, one sees shrunken patches, where in the midst of the red microscopically cavernous substance only little isolated portions of the white matter remain. The nutmeg appearance will then almost entirely have gone. Under these circumstances the organ is small, its surface uneven from the shrinking of parts, its capsule rather opaque, and often crowded with minute flocculent papilliform developments of the peritoneal textures. These little outgrowths are similar to the so-called Pacchionian bodies on the cerebral meninges, which, like them, are caused by chronic mechanical obstruction in the veins.

*Pigmental degeneration.*—Continental writers describe a pigmentary change in various organs as a result of long-continued exposure to malarial poisoning. A condition of melanæmia arises in consequence of the destruction of the red corpuscles of the blood in the spleen and the colouring matter escaping into the circulation. Consequently the brain, liver, kidney, and other parts become the depositories of this pigment, which is seen in the course of the capillaries. It is observed in the liver in the form of small granules in the portal capillaries, the lobules being surrounded by a black margin (see also Spleen). A pigmentary degeneration may, however, be sometimes found in other diseases than the malarious.

**Hæmorrhage.**—An excess of pressure on the blood-vessels from congestion may cause actual hæmorrhage into the tissue; we shall find, however, that this is due, in most cases, to a disease of the blood itself, as in purpura, where there is a disposition to bleeding from all parts. As, however, one very frequent cause of this purpuric state is hepatic disease, hæmorrhage into the tissue will be most frequently found in the morbid organ, and if with this there be a diseased heart, we have present a twofold cause for this condition. For in cases of morbus cordis we generally have a highly congested liver, the organ being in the state called "nutmeg liver," and sometimes (but more especially if the liver itself be diseased) we shall find blood actually extravasated into the tissue, as we have just now said.

In order to prove that this is the case, we have simply to take the end of the water-pipe and insert it into the portal vein, when in a few minutes a stream of water will permeate the whole parenchyma of the liver, passing through the lobules into the hepatic veins, whence it may be seen coming out into the vena cava, at first red, but afterwards colourless. If now a section of the liver be made, it will be seen to be quite white, except in those parts where hæmorrhage into the tissue has occurred.

**Fatty Liver.**—We have already said that fat enters into the composition of "nutmeg liver." It is also very frequent in cirrhosis, &c.

But the condition called fatty liver is that in which the whole organ is loaded with fat, this being its only or principal disease. A lesser degree of the same state can scarcely be called morbid, if we judge from the fact of fat being a normal element in the livers of many of the inferior animals; and also from the discovery of so much fat in the livers of persons who have died from some accidental causes, that we must suppose a great variety in the amount of fat may be constantly occurring. Thus we have inspected the body of a butcher, who was killed by an ox, dying an hour after his injuries. In him we found a liver so fat that it floated on water, and yet he was thought by his friends to be in good health at the time of the accident. The proportion of fat in the healthy organ is only 2 or 3 per cent.; but what excess above this constitutes a morbid state we cannot say.

In a healthy liver we find this fat forming small granules in the hepatic cell; if it be in excess, it forms globules, and in extreme cases the whole cell is filled with oil. In such instances the organ is much spread out and softened, and is of a pale colour, the edges being rounded as if the tissue were crammed to excess with the adventitious matter; at the same time it is of light specific gravity, so that the liver may float when placed in water. Lesser degrees are constantly met with, which we recognise after some experience by the appearance and touch; but there are various tests by which we discover the presence of fat: thus, the old and rough method, one, indeed, which we still sometimes employ, is to burn a piece of the liver; we hold it in the lamp a sufficient length of time to allow the water to be driven out; while this is taking place, if fat be present, blue sparks are given off, and after a time the piece burns, and the oil drops from it, and may be collected on a piece of paper. We may also use chemical means: placing a piece of the organ in a tube with some hot ether, and then pouring the fluid out to solidify. But the best test is the microscope, for by this means we may tell to a great nicety the relative amount of fat present, and whether it predominates in one part more than another; we may find, for example, small fat-globules in one cell and large ones in another, until we discover some entirely filled with oil. Fat may appear to be free in the tissue: this is usually from breaking of the cells in preparation; but we have seen fat undoubtedly free in the liver texture.

In most cases of fatty liver which we examine we find that the degeneration into, or increase of, fat takes place on the circumference of the lobules, and thus, in viewing a section by transmitted light, a dark black margin is seen around each lobule; this is the rule and is almost invariable. We can hardly enumerate the conditions in which fatty liver may be met with. In phthisis we know it is frequently seen; so also it is often associated with scrofulous affections of other organs; and we think it may be said that in most long-standing diseases, especially when occurring in persons who have been long bedridden, a fatty liver may be expected. It may also be looked for in those who have too much indulged their appetites at table, and especially in men who have drunk largely of beer. Generally a fatty liver is considered to be secondary to other ailments, or at most associated with them in connection with a common cause, and, indeed, it still remains to be decided how far, when a fatty liver appears to be the most marked morbid condition of the body, it may be looked upon as the primary disorder. We have already alluded to it as a consequence of phosphorus poisoning.

**Lardaceous, Waxy, or Amyloid Liver.**—This peculiar morbid condition appears formerly to have been confounded with that of fatty change, and the more easily as it is sometimes associated with it and occurs in the same class of subjects—the cachectic and the tuberculous.

The whole organ becomes more or less infiltrated with a peculiar translucent substance beginning in the smaller arteries, and afterwards affecting the capillaries and cells. The chemical nature of this substance is still doubtful since it is impossible to separate it from the tissue in which it is formed. No solvent has yet been found for it, and it is very callous to all chemical reagents.*

Dr. Dickinson says that dealkalised fibrin—that is, fibrin obtained from an acid solution—shows the same reactions as the lardaceous viscera, and he has constructod a theory of the formation of the disease on the foundation he thus lays down. Shortly, the theory is much as follows:—Firstly, those people who have a lardaceous viscera mostly have been the subjects of long-standing purulent discharge; secondly, pus is alkaline, and so carries off much alkali; hence he thinks we may believe that the long-enduring discharge of pus would carry off alkali enough to dealkalise some of the fibrin in the body, and that this fibrin is then deposited as lardaceous matter. He proposes to call the disease *depurative*, on account of the loss of pus which he believes to occasion it. The theory is very ingenious, but unsatisfactory in many ways; for in some of these cases no discharge of pus has existed; and, further, it throws no light on the curious distribution of the diseased material which is practically limited to certain digesting viscera, the kidneys, and the lymphatic glands; parts which, we shall see, have no direct functional connection. Lardaceous matter has a curious attraction for iodine, which produces in it a deep red stain. This red colour has been said in some instances, when seen under the microscope, to change to blue on the addition of sulphuric acid. This reaction closely resembles that of certain starchy matters, and has therefore suggested the term *amyloid*. Whatever this lardaceous or amyloid matter may be, no doubt it is in no way at all like scrofulous or tuberculous products, for these consist of new-formed cellular elements; whereas in lardaceous disease there are no new cells, but the morbid substance is deposited within the existing elements of the tissue.

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* See "Report of Committee of Pathological Society" in 'Transactions,' vol. xxii, and "Discussion," vol. xxx.

As yet the microscope has revealed nearly all that we accurately know about the amyloid substance; it shows us its site in the tissues, and also its peculiarities of appearance; for those elements of the tissue which are diseased are seen to be swollen with a highly refractile, clear, lustrous, resin-like appearance. But the outlines of the elements affected are obscured through the great refractability of the amyloid material.

A most interesting relation to the small arteries is seen; the inner coats of these are so affected that the arteries look like clear translucent tubes. The muscle-cells in the wall of the artery are certainly involved, and, no doubt, the lining membrane also; at least, this is probable, because the disease extends on into the capillaries, producing in them the same swollen lustrous appearance. On the other hand, the large arteries are never affected.

It appears that the amyloid matter extends from the vessels into the proper secreting cells of the liver, altering them in the same way to lustrous translucent little bodies, in which no nucleus can be seen. But the change does not affect all parts of the hepatic lobule together; it begins at those parts into which the hepatic artery more immediately enters. This is usually in the middle zone of the lobule, neither at its centre nor at its circumference. By comparing this with what we find in fatty and pigmented livers, we see that as fat usually occupies the outer zone, and excess of pigment is found chiefly at the centre, these three kinds of morbid deposit have their several especial seats, thus revealing a difference in the habit of these tracts in the lobule, though all parts of it have the same appearance when healthy.

It is in the hepatic artery that the first change is seen. The coats become swollen and the capillaries lose their distinctness. As the new material increases the cells disappear, and nothing but a little areolar tissue is seen. This is probably merely the remnant of the original structure, although in some portions of the liver the tissue is so marked that it looks as if a cirrhotic process might have been progressing at the same time. It would thus resemble a similar condition of kidney.

We must next observe that though the distribution of disease in the liver is generally uniform, or nearly so, yet sometimes it is partial and sometimes very unequal, so that portions of the organ show a yellowish-white colour, and may resemble at first sight syphilitic patches; but we have never seen any shrivelling result from this. Lardaceous disease enlarges the part affected, and we think it does not lead to shrinking, nor to a cicatricial appearance, as some have said.

The liver may be more or less infiltrated with the adventitious matter; when slightly so, the microscope may be required to recognise it; but when the quantity is excessive the liver becomes enormously enlarged, almost filling the abdomen, and acquires great weight; thus, a short time ago a liver of this kind weighed fourteen pounds. It is dense and very inelastic. Unless fat be also present in it, as it often is, the liver has its usual shape and sharpness of edge, but if there is much fat the edges are rounded. The organ, however, tends to mould itself upon the solid organs it touches, the posterior part being hollowed for the kidney; and if, as is often the case, it has reached far to the left side, there may be also a concavity for the spleen. Instead of feeling soft, it is hard and firm, and the peculiar sensation given to the knife when cutting it is very characteristic, like that of raw bacon, whence its name. Thus, it cuts firm, so that the thinnest slices may be taken off with ease, and if these be held to the light they will be found translucent, much like a tissue that has been placed in glycerine. We shall be able clearly to make out the lobules of such a lardaceous liver; we know that these lobules do not, as in some of the lower animals, form really distinct portions of the tissue, but are appearances due merely to the mode of distribution and connection of the blood-vessels; they are, however, remarkably well defined in the lardaceous organ. The tissue is peculiarly dry, and on pressure only a little watery pink blood escapes from the vessels. If the organ be only partially infiltrated, so that it is not yet evidently enlarged, the disease may be recognised by the increased weight and inelastic hardness given it; thus, so different is it from the fatty liver we met with the other day, which floated in water, that portions of lardaceous liver we have found to be of a specific gravity of 1084. In one case the right lobe was adherent to the diaphragm, and the lardaceous matter had so destroyed the tissue of the surface as to form a white layer almost an inch thick.

These lardaceous livers are found mostly in scrofulous persons, and especially in the young who have suffered a long time with disease of the bones, whence it has been suggested whether any gelatiniform material might have been taken up from the osseous system and carried through the body. But the bone itself in these cases does not give the amyloid reaction, so there is little foundation for that belief. Phthisis is another frequent cause, but only chronic phthisis, such as is called "fibroid." Indeed, any form of prolonged suppuration may bring it on. Twice we have seen it from old carbuncles; once from leprosy, and occasionally from cancerous sores. Syphilis without any suppuration will certainly cause it, so that it is not brought about exclusively by discharge of pus. The cases in which it has been set down to intermittent fevers appear all to have been complicated with syphilis. For further particulars as to lardaceous disease, see "General Diseases."

**Inflammation.**—In speaking of acute yellow atrophy of the liver, we have mentioned that the shrinking of the liver in yellow and red atrophy is of an irritant kind, but that it does not usually produce inflammation of the capsule or other signs of thorough inflammation. In these conditions the inflammation is so held under restraint, as it were, by the specific nature of the disease, that it does not reach the conditions proper to a characteristic degree of inflammatory disorder. The inflammations of the liver, as usually recognised, are acute or chronic. The only certainly recognised acute inflammation of the tissue of the liver in this country is that which leads to suppuration, and which is commonly called abscess of the liver. We have no anatomical evidence of any state of liver corresponding to acute Bright's disease in the kidney, or acute pneumonia in the lung, and this fact suggests that we may be overlooking important diseases of the liver.

*Irritative swelling.*—We must not, however, fail to notice the state of the liver in grave febrile disease, such as typhus, pyæmia, &c. In these diseases all the organs are bloated and soft, and loaded with blood, and the liver partakes in these changes very largely. It is easy to let this condition of the liver pass for a secondary state due to febrile blood-change, in the same way as we so regard the softened spleen; but some observers are disposed to give these livers more particular significance, and, in short, consider them to be truly inflamed. The state of liver to which we are alluding shows itself in a moderate swelling of the organ with a loss of its natural consistence, so that it is more pliable and soft and less elastic than is natural. The section-face generally has a dull shine, like cut moist clay; it is paler than natural; in fact, its look is often quite remarkable. The microscopic appearances are not so decidedly abnormal as the above characters might lead us to expect. Considerable changes have been described by some. Thus it is said that the liver-cells are swollen and granular, being, in short, in the condition termed by Virchow "cloudy swelling;" but this is a state not easy to recognise with certainty in a liver, as its cells are naturally cloudy and variable. It is further said that exudation-cells can be seen about the portal veins, and in the lobule-tissue near them. This, however, is certainly not a constant appearance. What is most constant is an evident enlargement of the lobules, explaining the swelling which the naked eye already sees; and further, there is evidently a soaked or œdematous swollen appearance of all the interstitial connective substances.

Before, however, we can include this condition among true inflammations we should require some evidence to prove a tendency on its part to run on into more certain inflammation. Instead of this, however, the capsule of such a liver is never inflamed, and no evidence has been brought to show that prolonged or severe cases of fever lead into inflammatory or hepatic changes, such as abscess or cirrhosis of liver. Hence the word irritation appears better to express what we know of it, or perhaps *granular degeneration.* Meantime this condition is no doubt very important among the lesions which bring dangers in fevers, and a better knowledge of it is much to be desired.

*Acute hepatitis.*—Inflammation may be chronic or acute; we know little of hepatitis in its acute form in this country, except in its results as abscess, and it is questionable whether it ever occurs as an idiopathic disease in a temperate climate. In cases of suppuration the adjacent tissue may be found in various stages of inflammation, and a hepatitis, we believe, exists during the early pyrexia, and precedes the formation of pus; but this stage is short, and is rarely seen, perhaps never in this country, as an *idiopathic* affection. In tropical climates, however, such a hepatitis is a common disease, leading to various results, as abscess or gangrene; the former may be cured. Or the inflammation, in the tropics, may be more chronic, and lead to cirrhosis.

*Abscess.*—This is occasionally met with arising from injury; thus a blow producing fracture of the ribs on the right side we have seen cause an hepatic abscess, and more than once produce a suppuration between the liver and diaphragm, which involved the former.

But hepatic abscess usually arises from pyæmia, either when this is of a general character, but much more often from a local pyæmia affecting the portal veins in connection with dysentery. In *general pyæmia*, as in the instance where death follows an amputation, abscesses may form in any part of the body. As a rule, these are confined to the lungs, and only in exceptional cases is the liver affected. We have, however, very repeatedly seen large abscesses in the liver, which sometimes have produced fatal peritonitis by bursting. It was stated by Pott, and since repeated, that such abscesses are more frequent after injuries of the head, but this we cannot corroborate from memory and without searching through the records, although we think it is true. It is thought that some of the elements of pus or putrid matter are taken into a vein and carried to the lungs, where they produce a peculiar form of lobular inflammation, which we have already described; and that if any of these elements should be carried into the general circulation, the other organs, as the liver, may be involved. The difficulty in understanding this process is, that the liver sometimes may be the only organ affected, or the suppuration is evidently older than that in the lung; nor is it further explained on the supposition that the infection is due to microbes. We have, however, only met with two or three instances of this; in one, it was probable that a blow had been received over the liver; and in another there was suppuration in the pelvis, which might have given an independent source for the purulent infection.

Besides these cases, arising from general pyæmia and injury there is another class which is associated with dysentery, and which is supposed to be dependent on the intestinal disease, or a *local pyæmia*. Dr. Budd observed that the majority of hepatic abscesses we meet with are in the persons of sailors who have been in the tropics, and in whom on post-mortem examination old ulcers are found in the colon, from which fact he was lead to believe that a morbid material had been taken up from the ulcers by the portal vein, and had set up inflammation and abscess in the liver.

We believe that the observations of Dr. Budd are correct, since our own and other records corroborate them. These show that, excepting those cases of hepatic abscess which stand in connection with the two causes just named, nearly all the examples of that condition are in persons from the tropics, and are associated with dysentery, for on looking through our cases, we find only one of abscess in the liver which can be designated by the name of idiopathic, and even here there was the history of a blow preceding the symptoms. But in these cases, although the liver and the colon are affected it is a question which affection stands to the other in the position of cause, and which of effect. The difficulty sometimes arises through the apparently recent state of the colic ulceration as compared with the chronic nature of the abscess in the liver, but more especially through the fact of the two occurring together only under certain circumstances. Thus no affection is more common in this country than ulceration of the intestine, and yet it is rarely followed by hepatic abscess. Moreover, in some parts of Europe a true dysentery occurs, and yet no hepatic affection with it. Thus, a few years ago several hundred bodies were examined at Prague, after death by dysentery, and yet in not one was the liver affected. Also in Norway a severe epidemic occurred with many deaths, and the only sequelæ observed were ascites and acute rheumatism. It is chiefly in tropical climates where hepatitis occurs that the connection is found, and therefore it seems at first more rational to attribute the hepatitis to some other cause than to the intestinal affection. Thus, in India it has been observed by our own surgeons, as well as by the French in Algeria, that during certain epidemics a number of soldiers will have dysentery, some hepatic abscess, and others the two together; in the latter case the doctors seem more inclined to attribute the colitis to the hepatic abscess than *vice versâ.* We believe, however, the fact still remains, that in persons coming from abroad with hepatic abscess, an ulcerated colon is almost sure to be discovered, or the cicatrices of ulcers; at least, our own experience in this respect corroborates Dr. Budd's. As proving the intimate relation between the two diseases, a case was shown at the Pathological Society by Dr. C. Turner, where a man died of hepatic abscess from an injury and where the intestines were found to be much ulcerated.

Also it must be said that the experience given by those who have met with numerous cases of hepatic abscess in the tropics differs somewhat from Dr. Budd's statement. Thus as in many as one fourth of 204 cases no traces of ulceration could be found in the intestine, though the patient had died of hepatic abscess. And Mr. Waring, who adduces these cases, states, as another important contradiction to Dr. Budd's view, that of 300 cases of hepatic abscess which proved fatal in India little more than one fourth had been preceded by symptoms of dysentery.

But we must remember, in estimating the value of evidence from such large series of cases, that the non-discovery of an ulcer is not a positive but a negative result, and it is surely probable that some inspections out of so many would be made hastily or under unfavorable circumstances, and scars of ulcer in the bowel are easily overlooked, while the whole of the tract that might furnish place for an ulcer is extremely long, requiring much more than average care for a thorough search. Hence we think that the non-discovery of ulceration in one fourth only of so many cases is by no means very trustworthy and conclusive proof of their non-existence. An infective inflammation, indeed, might occur without any ulceration. The most important positive evidence adduced against Dr. Budd's view appears to us to be that brought by Dr. Murchison, who points out a difference in the size and number of the liver abscesses which occur abroad and at home. Tropical abscesses are generally single and large, many abscesses in a liver being decidedly rare, while pyæmic abscesses, as we meet them at home, for the most part are small, and many are present scattered about in the organ close to its surface. Dr. Murchison's own experience has enabled him to speak with a knowledge of both the sorts of abscess he describes. We must not forget, however, that common pyæmic abscess of the liver in England, being part of a *general* pyæmia, is comparatively quickly fatal, so that the abscesses have little time to enlarge and break into each other. These abscesses, further, are prone to occur in the hinder part of the right lobe, the part which lies lowest when the patient is on his back. And in cases of some standing we have seen them breaking into each other as though a large collection would have in time resulted. Tropical abscesses, on the other hand, generally run a longer course in which to enlarge, and to unite if originally multiple; they also by preference affect the hinder part of the right lobe. In fact, the liver-abscess of this country which occurs in pyæmia is generally overlooked during the life of the patient, whose troubles from co-existent abscesses in the lungs, joints, &c., together making up the complex which we call pyæmia, reduce the hepatic suppuration to the dimensions of a weighty addition to an already desperate evil. We have never known abscess of the liver resulting from general pyæmia in this country to have so much time allowed it, or to be so far the solitary mischief as to enable it to become an object for special treatment. Herein arises a great and most important practical distinction from the tropical abscess, which may become chronic because it often is the only very grave disease present. Indeed, it may when very large be wonderfully secret; no pain and scarce any hectic disturbing the patient. These large chronic tropical abscesses are those which afford opportunities for successful treatment, and in some 20 per cent. allow of cure. Generally they are single, and rarely more than two are present. Occasionally, a large chronic abscess is found in connection with smaller ones scattered through the organ. The latter are then recent.

Next let us pass on to consider the stages of formation to be observed in hepatic abscesses. These are, of course, best known in the pyæmic abscess, having been studied in this country and in Germany by professed pathologists, and with much care. The opinion regarding them which up to the present has prevailed among pathologists is sufficiently expressed by the name "embolic," which has been given them. The history of the process on the embolic theory is engagingly simple; it can be understood on purely mechanical considerations. Shortly it is this: A portion of clot from some vein in the primary ulcer, the source of the mischief, is carried into a branch of the portal vein, where it lodges and cuts off the blood supply from a certain area corresponding to the part the starved portion should supply. The capillaries on the confines of the territories around dilate; but yet though they bring blood into the starved part the natural *vis a tergo* which should come down the obstructed vessel is stopped by the clot, and hence the blood in these dilated capillaries stagnates. Herein arises the first stage of congestion characterised by great engorgement of the affected patch of liver, especially of its circumferential part. According to the latest theories of infection there must be a transmission of microbes in order to produce septicæmia or pyæmia. A thrombosis is thus caused and the processes go on as we shall describe.

The next stage in the process has been explained in the following simple form; the obstructed territory of the liver cut off from its natural circulation of blood, perishes, undergoing separation from the part around, the disintegrated portion lies in the cavity formed by its separation, mixed with pus secreted by the surrounding wall of living tissue, which in sufficient time is converted into the usual abscess wall or "pyogenic membrane." It will be perceived that this description corresponds exactly with what is stated to occur in the lung also through embolism; but we have already shown in the case of the lung that clots obstructing the artery do not lead to such death of the obstructed part, unless the plug comes from some severely inflamed part. There is plenty of evidence to afford ample proof that mechanical obstruction of the portal veins will not cause death of the obstructed portion. Thus we have several times seen branches of the portal veins stuffed with cancer brought in from cancer of the stomach or intestine, and we have also seen all or part of the portal vein entirely obstructed in cases of cirrhosis, but in these cases no trace of any approach to death of the obstructed part occurred. Hence we think that in the liver, as in the lung, embolism of the supplying vessels does not lead to abscess through simply mechanical consequences, but rather that the irritant quality residing in the clot tfrom the inflamed region and belonging to it as part of the products of inflammation there, or the presence of bacteria, forms the determining cause of the suppuration that arises when such a clot enters the liver. Still we must not leave the mechanical effects out of consideration; it cannot but arise that the dilatation of the vessels with stagnation of their contents must compress and weaken the liver tissue, and prepare it to yield to the destructive influence of the inflammatory cause.

The second stage shows a breaking down of the portion involved, with every appearance of rapid disintegration into a purulent fluid. We must, however, note that there is usually no sign of gangrene.

In the next stage the abscess completes itself and may discharge its contents by bursting. This occurrence in pyæmic or embolic abscess is rare, death coming early and even usually before the abscess has become rounded or acquired a smooth interior, so that we find it enclosed by the more or less ragged remains of the doomed tissue, as yet not entirely destroyed. If we examine the veins of the liver around the acute abscesses, we often find that the pus has entered a portal or hepatic vein to some length; and may even thus extend into the right heart.

The large abscesses which Dr. Murchison calls tropical, and which are found in those who come from hot climates, are of older standing, so that they are generally more or less encysted; that is, the wall of the abscess is tough and fibrous, like a special cyst enclosing the pus; the cyst arising through a thickening of the natural fibrous tissue present in the implicated part of the liver. The colour of the contents varies, being greenish-cream colour when pure, but often getting various tints of yellow or brown through mixture with bile or altered blood. Such colours of the pus we should be careful to remember, for we have known the character of the pus lead to correct diagnosis when an abscess of the liver has burst through the diaphragm into the lung and been spat up; patients have sometimes complained of the bitter taste of such pus.

It is important to be aware of the various modes of termination in different examples of tropical abscess of the liver. They may exhaust the patient, and kill without reaching the surface; and they may diminish and shrivel, the cyst contracting around them, until only a pasty, or putty-like, or white dry matter is left. A liver in the Museum shows a cheesy mass surrounded by firm fibrous tissue, and running in the course of the portal veins; other portions of the liver are lardaceous. The interior of the colon is covered with old cicatrices. (Taken from the body of a soldier long resident in the tropics.) But the more ordinary course of such abscesses is to discharge themselves within a few months of their first formation, the direction in which the matter escapes depending on the position it occupies. It may go into any of the cavities to which the liver is adjacent, such as the pleura, pericardium, colon, stomach, duodenum, gall-bladder, or even the pelvis of the kidney. To reach any of these, except the last two, the peritoneal surfaces must become adherent prior to the perforation. Should such adhesion not occur, the abscess may burst into the peritoneum. Or passing through the diaphragm it may find the lung unadherent, so bursting into the pleural cavity; or the inflammation may cause adhesion of the lung, when the abscess bursts into the lung tissue and reaches the bronchial tube. As the abscesses are usually in the right lobe and near the surface, it will easily be inferred that the most frequent way of escape is through the diaphragm upwards, or to the surface of the body outwards, seeing that by far the larger part of the right lobe is separated by the diaphragm from the lung and parietes. Hence these ways of escape are the most frequent; but we must remember that when the abscess comes through the abdominal wall, which is the most favorable course it can pursue, the fasciæ and the tendons cause it to take an irregular and often a long course, burrowing in the substance of the abdominal wall, and making its appearance at some distance from the real seat of the abscess. The surgeon, however, usually is called to interfere, and makes the straightest way for it he can.

While speaking of abscess in the tissue of the liver, we must bear in mind that a hydatid cyst may suppurate, so forming an abscess in which, of course, the bodies of the hydatids will be seen, unless the case has lasted so long that they are disintegrated. Some long-standing abscesses may, perhaps, be of this nature.

Besides the pyæmic abscesses due to embolism, and the tropical abscesses which fall doubtfully under the same kind, there are other suppurations in which the liver is concerned, though by far more rarely, especially *suppuration of the portal vein*, hepatic duct, and abscess outside upon the liver, or "*perihepatic abscess.*"

*Suppuration of the portal vein.*—Sometimes this takes a very definite form; the portal vein-trunk and its branches being bathed in pus, and their channel obstructed with blood-clot, which is more or less altered, and generally contains pus; or the channel may be open, for a greater or less extent, into the suppurating space around. This is not a frequent form of disease, but many cases are recorded; some ulceration or suppuration in the field of origin of the portal system will be nearly always found to have given risen to the portal suppuration, or *pylephlebitis*, as it is called. Thus, in a case we had recently undar observation, we found abscesses in the submucous tissue of the rectum, which so implicated the hæmorrhoidal veins, and extended up the inferior mesenteric vein as to reach the portal vein, around whose trunk there was extensive suppuration, spreading irregularly into the liver along its branches. The disease which gives rise to such suppuration may be in the alimentary canal, gall-bladder, or spleen; the portal suppuration arising through an extension of this inflammation along the wall of the implicated branch, and so differing from embolic abscess of the liver in this, that the latter is due to the conveyance of the cause of suppuration by the blood-stream, and not by the vein wall. Both kinds of suppuration, however, as we see, are generally evidently due to disease in the field of origin of the portal vessels. A fish-bone penetrating, or an abscess of the spleen reaching the branches of the portal vein, has been known to excite this suppuration.

Dr. Carrington has collected a considerable number of cases of diffused suppuration through the liver where the cause was not apparent. In many there was an abscess in Glisson's capsule and around the pancreas, but no special lesion of this organ perceptible. He suggested the possibility of primary suppurative inflammation of Glisson's capsule. ('Guy's Hospital Reports,' vol. xxvi.)

We must further notice, though it is perhaps too obvious to need remark, that all large abscesses of the liver, destroying as they do great areas of its tissue, must cut across more or less considerable branches of the portal vein, as, indeed, of the hepatic vein also. It is interesting to observe how the vein so cut across is preserved from the entry of pus. We generally find that the suppuration does run some little distance along the hepatic vein, and also, though less frequently, along the portal vein. The implicated parts of the vessels are occupied by pus from the abscess, but this is cut off from the blood by a layer of yellowish-coloured clot or fibrin, which has a smooth convex surface toward the blood. A similar clot lines the diseased part of the vein, and closes its lateral branches. In this way tolerable security is reached. Another source of suppuration in the gall-blandder and ducts will presently be mentioned.

*Suppuration about the liver.*—We have seen several cases of large abscesses between the liver and diaphragm, or between the liver and stomach; the liver tissue being only compressed by, and not involved in, the abscesses, which lay quite outside of it. Some of these were traced to injuries, but for others no cause could be assigned. In one case two abscesses about the liver were associated with an abscess on the outside of the spleen, and there was, in the case of either organ, an older plastic inflammation over the whole surface, firmly uniting the organ to its bed, and favouring the insulation of the pus. It appears that this was an extreme form of that more chronic perihepatic inflammation which results from spirituous liquors in not a few cases. It is possible that such abscesses may arise by suppuration of cysts in this region. We have twice clinically met with large cysts here in male patients; the cyst-contents being an albuminous serum, once without any blood, and once with little. Both cases, fortunately, recovered after tapping, but suppuration of such a cyst would explain perihepatic abscess.

*Chronic inflammation.*—Under this head we include those changes which result mainly in the formation of fibrous tissue; suppuration, although it may be of long standing, we have preferred to speak of under the acute changes.

*Indurative hepatitis* or *cirrhosis* is the most important disease to which the liver is liable in this country. It is generally described as an inflammation of Glisson's capsule, whereby lymph is first thrown out around the portal vein, and subsequently contracting, causes a very general shrinking and hardening of the organ. It cannot, however, be said to be generally true that the liver is in the first place enlarged and subsequently shrinks. In the majority of cases of cirrhosis the liver remains larger than natural.

But in some cases there is no proof that the lymph was ever produced in quantity as in active inflammation; rather it appears certain that the whole change is chronic; sometimes, however, where the capsule is much thickened, and the organ is closely adherent to surrounding parts, the term hepatitis may with more propriety be used.

Cirrhosis is, however, in most cases essentially a slow or chronic affection, and an idea may be formed of its nature by the supposition of a tough fibrous tissue being diffused through the organ in the course of the vessels, which tissue contracting squeezes the parenchyma into a number of nodules. It might be thought that, as a consequence of this, the organ would become hard, and at the same time small, the surface irregular, and covered with projections, or "hobnails," as they are sometimes called. This is the condition we see on the surface, and on a section we observe similar rounded nodular masses, separated by a new tough adventitious tissue. In some instances the knobs are very large, in others they are small and diffused through the whole organ, the surface remaining nearly smooth, so that this form merely shows a granular surface; hence the name "granular liver" has been given to it. It has been stated that the granules vary in size according to the kind of spirit which has caused the disease; but of this we have no knowledge. The explanation of the difference lies in this, that in some the larger, in others the smaller vessels are especially affected; thus smaller or greater portions of tissue are free between those affected vessels whose contracted and thickened courses form the fibrous seams, while the intermediate free tissue makes the hobnails or granules.

Another cause of variation in the size of the granules is due to the presence or absence of fat in them; the fat, when present, enlarging them considerably. This fat is usually found in the more corpulent bodies of beer-drinkers, while in the wasted frames of those who have suffered from spirit-drinking the liver is often darker, smaller, and apparently devoid of fat. On a section of the cirrhosed liver, then, little or none of the natural appearances can be seen. The colour is usually much, though very variously, altered; opaque, whitish yellow, bright yellow, or dark greenish, or brownish colours prevailing; the first due to fat, the other three colours to bile-pigment of different hues, and in varying quantities. There is commonly a prevalence of unusually bright yellow, from which, indeed, the name is derived; Laennec, who gave the name, erroneously supposing the yellow matter to be an adventitious diseased product. This is not the case; on the contrary, the soft yellow or variously coloured substance is the remains of the hepatic tissue, not easily recognisable, as the characteristic regular minute foliaceous-outlined lobules are not to be seen, but instead of them the soft substance forms coarse, roundish, unequal islets, embedded in tough, fibrous intermediate matter. The quantity of this may become very great, so that one fifth or one third may be composed of fibre, and that even when the liver is above its normal weight. This fact proves the error of those who would deny that the fibrous substance of the liver augments in cirrhosis, describing the change as mere wasting of the cell-elements of the lobules, which, of course, would give only a relative and not an absolute excess of fibre. We have already seen in chronic atrophy that the wasting of the cells leads to no such result, and the enormous proportion of fibrous substance in an enlarged cirrhosed liver places the fact of its excess beyond doubt. Occasionally parts have a worm-eaten appearance or that of minute cysts.

Hitherto we have spoken of this substance as fibrous tissue. Microscopic examination, however, shows in it elements responding to its active and encroaching nature; it is crowded more or less with small cells. It is often full of networks of biliary ducts, which have led to the supposition that in it an effort arises to restore the hepatic tissue again by the same outgrowth from the ducts as originally produced the hepatic tissue; further, it is loaded with large vessels, easily injected from the hepatic artery, for the compression exerted by the new formation tells more upon the feebler current in the portal vein, so that the hepatic artery enlarges to compensate.

The *histology* of this disease shows it to be essentially a growth of connective tissue around the portal vessels, and in which there may sometimes be produced new blood-vessels and hepatic canals. The size of the nodules varies according to the size of the portal vessels involved, and in consequence we may have the nodular, granular, or smooth variety of cirrhosis or these passing into one another. Drs. Creighton, Hamilton, and others, have been proving how the new connective tissue is derived from the hepatic cell. The latter pathologist exhibits drawings of the natural cells becoming swollen, the nucleus undergoing segmentation, two cells being produced, and the cell becoming spindle shaped, the latter soon showing itself as a bundle of fibres until the process is complete. It had been the opinion of other pathologists that the blood-vessels or bile-ducts had been the source of the new tissue. In early cirrhosis if a section be made of the liver an abundance of nuclei is seen around the portal vessels. In more advanced stages we find the new tissue full of small canals which can be injected from the hepatic artery, and very often there are seen rows of cells resembling new biliary ducts. When this is well marked it is thought that it constitutes another form of cirrhosis, a form where the connective tissue is found surrounding the ducts rather than the blood-vessels, the whole organ becoming enlarged. This will presently be described under the name of hypertrophic or biliary cirrhosis in distinction to the common cirrhosis. Charcot speaks of an intercellular cirrhosis where a new connective tissue is found between the lobules. This is sometimes met with especially in congenital syphilis. It is now many years ago since Lionel Beale described the formation of new blood-vessels in the connective tissue. These have generally been regarded as hepatic arteries, whilst the hepatic veins have shrunken or disappeared.

We need scarcely say that cirrhosis of the liver is often found complicated with its other diseased states; especially with syphilitic changes, lardaceous disease, the nutmeg state in heart disease, and, as we have just now said, the various degrees of fatty change. In a pure case of cirrhosis the capsule of the liver is not thickened much, but some examples will be found coated with a more or less, and often very, thick capsule of adventitious tissue; this is always readily separable from the liver, leaving its proper capsule still on it. Such a coat exercises an immense compression upon the liver, flattening the gall-bladder and rounding the edges in a remarkable way, often by bending them over, so that when the coat is stripped off, the edge of the liver is found bent down upon the under surface of the organ, from which it rises when the false membrane is raised. A further proof of the tension exerted by this membrane is curiously given by its own appearance, which is always areolated, round holes extending more or less deeply into its substance. These are gaps which it has torn in itself during its contraction. When such a membrane exists upon the liver, one like it is generally found upon the spleen also.

It has been suggested as a possibility that cirrhosis of the liver sometimes commences by extension inwards of an inflammatory thickening of the capsule, such as we have described. In the case of the lung such an extension from a thickened pleura is believed to occur, so that chronic pleurisy leads to induration of the lung. That such a process outside the liver would cause a contraction and an impeded flow of blood through it sufficient to give rise to dropsy there can be no doubt; but as to whether it may actually involve the tissue to produce cirrhosis there is more uncertainty. It is just possible, however, that here may lie the explanation of those cases of cirrhosis which sometimes occur in young and temperate persons; that the affection has commenced as a simple peritoneal inflammation, and then the organ beneath has become involved, as may be seen in a drawing of a hobnail liver, taken from a girl eleven years of age, who died in this hospital a few years ago.

A considerable number of cases of cirrhosis have been observed in *children* where there was no possibility of alcohol having been the cause of the disease. In one of our last cases, under Dr. Taylor, there were well-marked hobnails with thickening of the capsule, and the microscopic examination showed a large quantity of fibrillated tissue containing nuclei together with newly-formed blood-vessels and biliary canaliculi. In some cases tubercle has been found in the peritoneum, and this has given rise to the question whether the cirrhosis was in any way connected with it; whether, indeed, the tubercles might afford the nuclei for the growth of a connective tissue which would be the starting-point for the cirrhosis. There is no proof whatever that this ever occurs. There is, however, a very hard liver sometimes met with owing to its infiltration with connective tissue and in which tubercles are scattered. Nor is there any good reason to think that cirrhosis may arise from chronic congestion, as we have already said when speaking of the so-called cardiac cirrhosis. Then, again, the possibility of its arising as a capsulitis has been considered as well as its origin in a malaria. Also the suggestion of Dr. Klein whether it might have its starting-point in an exanthem, as scarlatina, and a chronic affection of the liver be set up in the same way as a disease of the kidney in this affection. The late Dr. Budd thought it probable that some injurious products of digestion might be taken up by the portal vessels and so set up a similar morbid process as that which is more usually caused by alcohol; the modern doctrine of leucomaines may throw further light on this subject. The difficulty of tracing cirrhosis in children to the usual cause has tended to strengthen the belief that it may arise sometimes in the ducts of the liver giving rise to the so-called hypertrophic cirrhosis. This would be the view taken in the case of a little boy we are now seeing, who had enlargement of the liver accompanied by ascites, and in whom, after a time, the increase in size gradually disappeared. About two years before the enlargement was discovered he had jaundice.

One of the most interesting facts connected with cirrhosis is the production of a *compensatory or collateral circulation*. It is well known that in this disease we see large veins coursing over the abdomen, but it was not until recently demonstrated by Sappey why the systemic veins should be thus enlarged as a consequence of portal obstruction. It is found that if the portal vein be injected, the fluid will pass on to the surface of the liver, into the suspensory ligament, gall-bladder, and neighbouring parts, and not only through these channels but by the opening up of the old umbilical vein or some smaller veins which may be found running in the course of the round ligament. Dr. Goodhart has placed in the Museum an accessory vein as large as the portal vein itself. Communication then is made between it and the epigastric, and so brings the systemic and portal veins together as is seen in some of the lower animals.

*Local inflammatory or fibrous changes.*—These are not unfrequently met with, resulting from a local irritation or injury. Thus, in cases of gall-stones, when inflammation extends to adjacent parts, the liver may become involved, or, as in a case we met with lately of a man who for many years had suffered with pain in his side after being run over, a large mass of liver was converted into a fibrous tissue; and, indeed, cicatrices and results of chronic inflammation may be found in this organ as elsewhere. Local patches of thick fibrous tissue resembling cartilage are often met with on the surface.

In all cases, however, especially where more than one local fibrous scar-like patch exists, search should be made for other evidences of syphilitic taint in the body, and this with greater probability of success when the scars are on the surface and adherent to the adjacent parts. Multiple scars of this description almost always arise from syphilis.

*Hypertrophic cirrhosis or biliary hepatitis.*—A few years ago Cornil described a form of cirrhosis in which the liver was much enlarged from the overgrowth of connective tissue, and in which there was found a development of a network of interlobular biliary canaliculi. Charcot corroborated this statement, but other writers, whilst agreeing in the main, have shown that the development of canaliculi also takes place in many forms of liver disease, and it must be remembered that in atrophied portions of liver the natural ducts become much more evident to the eye. It is said that the patients who have been the subjects of this hypertrophic cirrhosis have generally had jaundice, and this is considered to strengthen the opinion that the disease has commenced in the bile-ducts. The liver has been said to be much larger and heavier than normal, of a dark colour, nodulated, and hard. As in ordinary cirrhosis bands of connective tissue are seen running through the substance, isolating portions of the tissue into nodules. On more minute examination it is said the characteristic tissue is not seen surrounding the portal veins, but around the biliary ducts, and that sections which display the portal vein, hepatic artery, and duct make the thickened condition of the duct very noticeable, this being surrounded by a thick layer of connective tissue composed of bundles of fibres. It is said also that the parenchyma contained in the nodules has not undergone the same compression as in ordinary cirrhosis; the hepatic cells for the most part still retaining their size and character, and even if any are found shrunken they are still entire. The disease is said then to be characterised by an enlargement of the liver due to an inflammation of the hepatic ducts and canaliculi situated in the portal spaces, and that a large production of connective tissue takes place around them, but the tissue of the liver itself is little affected, the cells appearing little changed from the normal.

In favour of Charcot's views it may be said that both Beale and Legg found that after ligature of the hepatic ducts a new connective tissue commenced to grow around their branches. Before accepting these views it must be remembered that a new development of biliary canaliculi lined with epithelium has also been met with in ordinary cirrhosis and other forms of chronic hepatitis. Cornil had already described these canaliculi formed of large cylindrical cells in ordinary cirrhosis as well as other features, leaving no characteristic distinction between it and this hypertrophic form. Then also, as Dr. Goodhart has argued, obstruction of the ducts, followed by inflammation, is by no means uncommon, but the results on the liver spoken of are certainly not frequent; and then, as regards size, two thirds of all cases of cirrhosis are above the ordinary weight of the healthy organ.

It should also be remembered how difficult it is to prove the existence of the new growth of parts, seeing that the contraction of the connective tissue brings the ducts into undue prominence; and in using the term ducts it must be remarked that these are nothing more than a double row of cells with no definite walls. Long before the pathology of these cases was given, Dr. Habershon had stated his belief in the enlargement being due to hypertrophy, since there was no wasting of the cells, and therefore he presumed they existed in larger numbers. In these cases too jaundice had existed. This would be then a real hypertrophy of gland tissue.

It may here be remarked that not only is the nature of a nodular liver difficult to make out during life, but even when the organ is under the eye a most careful examination is required to unravel it. Dr. Fagge exhibited a specimen at the Pathological Society in which there were round, soft nodules, looking exactly like cancer, and this was his first opinion. He found, however, that the nodules were composed of hepatic cells and fat. On the other hand he showed a smooth, hard liver, having the appearance of cirrhosis, but which was really cancer. It is not uncommon, however, to find a cancerous and cirrhotic condition mixed.

**Syphilitic Fibroid Deposits, or Nodules.**—These deposits occur in the liver as well as elsewhere, in the same way as nodes appear on the bones, or as deposits of fibroid tissue in the muscles, or as similar deposits in the various other parenchymatous organs, as the brain, spleen, or testes. The leading character of these changes is their strict localisation; the part affected undergoing extensive changes, while the remainder of the organ is healthy. The outline of the liver is altered more or less by the true syphilitic deposit. Scar-like depressions mark the surface; they may be so numerous and deep as to divide the liver up into a cluster of irregular masses, to which the name "botyroid" has been given, from its likeness to a bunch of grapes; or fixing especially on one lobe or part of a lobe, the disease may entirely destroy it; the remainder, which as we have said is healthy, undergoing hypertrophy in compensation.

When a section is made through such a syphilitic scar the characteristic syphilitic gumma may be seen, but not always; for after an uncertain but usually long duration, the caseous gumma disappears, and nothing but fibrous tissue is left. The earlier stage of syphilitic action is rarely seen in the liver; it appears as a sharply localised, deeply reddened, swollen patch, the portal venules of which have a grey lymph around them. What we more usually see, however, is a later stage of the disease when there is a pale yellowish, cheese-like nodule of irregular outline, surrounded by a fibrous zone, the outer edge of which loses itself in the lobular tissue, the lobules dwindling gradually in its grasp This fibrous zone is never very broad; the change is, as we have said, strictly localised. The cheesy centre varies in consistence from a gristle-like toughness to a pulpy softness; it is sometimes mortar-like from cretaceous change.

Occasionally a gumma softens down and liquefies, so that a cyst may be found full of a flocculent matter consisting of the débris of the growth. In the case of a man who had a large fluctuating tumour over the liver, this was opened and a curdy fluid escaped. In another case a similar material burst into the bile-ducts; and in another a semi-fluid matter of a like kind was found in the mediastinum. It is known that nodes on the bones often liquefy before they disappear under the influence of medicine.

Besides these more characteristic nodular forms a more diffused fibroid change may be met with in the liver in the same manner as the fibrous streaking in the testis. It is often associated with a capsulitis, and this condition therefore is by no means easily to be distinguished from ordinary cirrhosis, and therefore the name syphilitic cirrhosis has been given to it. We not infrequently see gummata developing in a liver which is otherwise cirrhotic, and although we have no doubt as to the origin of the gummata we are uncertain whether to attribute the cirrhosis to syphilis or alcohol. The microscope will not assist us much as there is really nothing characteristic in the decaying fibroid and cellular elements, which are the only products found. The new tissue, however, seems to be an increase of the fibrous element of Glisson's capsule. The syphilitic affection is frequently found associated with the lardaceous, the latter being a condition peculiarly related to the later stages of syphilis.

*Infantile syphilis.*—The same disease which is found in adults may be met with in infants. Our Museum contains the liver of a child aged twelve years full of nodules, and a similar specimen was exhibited at the Pathological Society, from a child, aged nine years, and Dr. Barlow showed another from an infant only twelve weeks old. In other cases of infantile syphilis the organ is found merely uniformly enlarged as Gübler first described.

In our Museum specimen of this kind it was large, hard, resistant to the knife, and bloodless. It could not be broken by the finger except by using considerable force. It was occupied throughout by a newly-formed connective tissue and nuclei infiltrating the whole organ. Besides this perfectly homogeneous liver and the nodular one, some authors have described an infantile syphilitic liver as granular.

It must be remembered that sometimes we find livers of very great size in infants due to infiltration of cells or connective tissue, and in which there is no history of syphilis. When found in connection with rickets the term *albuminoid* infiltration has been given to them. Several examples of very large livers from infants have been shown from time to time at the Pathological Society, and of whose nature there seemed a doubt. They were occupied by round-cells or leucocytes, and new fibrous tissue; they had been styled syphilitic, leukæmic, and diffused lymphosarcoma.

Mr. Symonds more recently had the case of a syphilitic child, a month old, where the liver was large, smooth, and firm, and contained nucleated fibre. Cells and wavy fibre were scattered through the organ.

There are no doubt other changes in the liver to be occasionally met with under various circumstances; thus, in cases of poisoning by arsenic, when the liver has become affected through absorption of the poison, we have seen the organ present a peculiar appearance; in one, particularly, we remember it was pale like a fatty liver, and soft, with a pinkish hue in parts, and here and there an arborescent ecchymosis; this purpuric tendency in poisoning by arsenic we have already spoken of in its effect upon the heart. In xanthelasma Dr. Fagge has described a large granular liver somewhat resembling cirrhosis.

**Injury.**—Rupture of the liver is very common; but as it seldom occurs without a very severe fall or blow, forms often part only of an extensive fatal injury. As we before mentioned, the laceration of the abdominal organs mostly occurs by tearing, due to a stretching of the tissue; and thus, in the case of the liver, the most convex parts on its upper surface give way, although these are the most protected. If the injury be slight, the surface is fissured or cracked, just as can be artificially produced by violently bending the organ upon itself. If this be the only injury, and no great hæmorrhage occur, recovery may take place, for a laceration of the great organs in the body is by no means necessarily fatal. Thus, in a case which we examined some years ago, of a man who fell from a scaffold, injuring his head as well as his abdomen, and whose death occurred some weeks afterwards from cerebral disease, there was a long laceration in the liver quite healed; and as regards the blood which had escaped from it, this was seen to have produced a dark layer all over the peritoneum, a part having been absorbed, and a part remaining as a pigmental covering to the serous layer; there was, however, no inflammatory product. It is worthy of remark that in some of these cases large bile-ducts are torn through, and bile freely effused along with the blood, giving a deep yellow stain to the lacerated surface, and yet no inflammation is set up thereby. The torn surface of the liver is tolerant of its own secretion; even when the peritoneum was reached by this yellow stain, the acute peritonitis which bile would be expected to produce certainly did not occur in any of our cases. We have preparations and drawings, showing lacerations of the liver undergoing repair; and in a case we lately saw, where a bullet had penetrated the diaphragm and lodged in the liver, it appeared as if a cyst were about to form, to render its presence innocuous. When the liver is directly wounded, this is very often from fracture of the ribs; there is, of course, puncture of the diaphragm and thus often hæmorrhage into the chest. A specimen on our shelves shows a needle in the liver.

**Spontaneous Laceration.**—We have no well-authenticated case of spontaneous laceration; but it may be that such an accident has occurred, for it is remarkable how frequently we from time to time hear of instances of injuries to some of the abdominal organs arising from violent exertion. Thus we have had a case where a man made a violent effort to extricate himself from machinery, during which it appeared that he had ruptured the hepatic artery. At the same time we read of a girl who in like manner ruptured her gall-bladder. In a specimen of liver we possess in which there are several lacerations on the surface, it is said that death took place very speedily after violent vomiting; the inspection showed a large quantity of blood beneath the peritoneum which had proceeded from these rents in the organ. This reminds us of the stomach already shown which was said to have been rent during vomiting. We mention these cases merely to state that there is some amount of evidence, though it is slight, towards showing the possibility of spontaneous lacerations of the liver. On this subject, however, it is very important to remark that where the liver has been ruptured either by blows or by being run over, no sign whatever of the injury may be found either on the surface of the body or in the abdominal parietes. So that in no case of reputed spontaneous rupture can post-mortem evidence afford any proof of its spontaneity.

**Morbid Growths.**—The liver is very subject to these, like the lung, but each organ shows preference for particular tumours. It is rarely the seat of innocent or non-infectious tumours, while it is the favourite place for the formation of secondary cancerous growths of many kinds, especially of carcinoma proper.

*Adenoma.*—For a long time there had been observed circumscribed growths in the liver, and the nature of which was evidently not cancerous. These were mostly nodules of various sizes, and generally surrounded by a capsule. The microscope showed them to consist of material more or less resembling that of the hepatic tissue, being composed of tubules or spaces of various sizes and shapes lined with cells of polygonal or cylindrical character. Greenfield, who was one of the first to give a good description of these adenoid growths, says that the section under the microscope shows a striking resemblance to a section of the cortical part of the kidney. He styled it *primary columnar epithelioma* of the liver, and regarded it as a variety of cancer, as he had found it associated with a similar disease in the lung and elsewhere. Rindfleisch described the disease under the term *cylindrical-celled epithelioma.* The term adenoid is sufficiently expressive as denoting a growth which consists of columns of cells having a tubular arrangement radiating from a centre like the normal hepatic structure, and corresponding in this way with the adenoid tumour of the mammary gland. Dr. Mahomed and Dr. Paul discussed its nature at the Pathological Society. They were inclined to think that, besides the cylindrical form, they sometimes met with a growth which more closely resembled the liver structure; that is, an encapsulated tumour composed of cells orderly arranged around the central vein and containing bile-ducts. Such as these they regarded as primary, whilst the cylindrical form was often secondary. Dr. Paul proposed a division into hyperplastic adenomata and heteroplastic or tubular epithelioma; the one arising from the liver-cells, and the other from the epithelium of the ducts—therefore acinous and tubular. It is a question whether there is not in this way a gradual progression towards true cancer, which would quite accord with the doctrine that cancer-cells are converted hepatic cells.

*Cancer.*—The appearance of cancer of the liver varies much in different specimens, both according to the extent of development and the character of the disease. Sometimes the liver is of enormous weight, and yet only small patches of true liver tissue remain scattered through the substance. In such cases parts of the liver will occasionally be found in a very perfect nutmeg condition. This we have traced to an obstruction of the corresponding hepatic vein by pressure of the cancer-masses. The appearance of the cancer differs also in different cases, very much through this, that in some it forms bold tumours that push aside the tissue, while in other examples it alters the tissue without much displacing it, the lobular substance being swollen and changed to the colour of the cancer growth, but still retaining a great deal of its natural construction. This latter form is described as infiltration. The first kind, the expanding and compressing tumours, are usually rapidly-growing *medullary carcinoma* or *sarcoma.* One may ascribe the reason of the circumferential compression to the circumstance that the multiplication of the elements within the affected area is greater than the rate of infection of surrounding tissue at the growing margin, so that the increasing interior mass pushes forcibly upon the as yet unimplicated surrounding liver. But in the infiltrating kind, which are generally the slower-growing forms of scirrhus, often secondary to cancer of the breast, the spread of cancerous infection is more rapid than the increase in number of the cells in the infected area; hence there is no overgrowth and pressure, but the marginal increase keeps pace with the central, and the order of the infected parts is little disturbed. An infiltration, however, in a sense of a pouring in of cancer among the healthy tissues is not to be seen here or anywhere, for the cancer is not added to the tissue, but composed of the transformed tissue itself. Another feature of liver-cancer is the cupped appearance presented by the large nodules or masses that rise above the surface; this feature is the more common because so large a part of hepatic cancer-growth is found at and involving the surface. Why this should be we cannot say; but if we do not see cancer on the surface of a liver, we are nearly sure not to see it on section. The cupped form of the cancer is due partly to the degenerative changes which its older central portion always undergoes, for however unfortunately obstinate the vitality of a cancer this vitality is not due to any tenacity of life in its individual cells, which indeed are sufficiently frail, but too prolific; hence the central parts of cancer waste, and contrast with the peripheral part, having the yellowish, bloodless, cheese-like appearance usually ascribed to tubercle, which appearance was indeed formerly supposed to signify mixture of tubercle with the cancer. These cheesy patches show microscopically decayed and perishing structure, but of the same nature as the rest of the cancer, whose *necrobiosis*, as it is termed, or more shortly as we will say it, whose *senescence* it indicates. Besides the natural senescence of the cancer-cells, other influences work to produce the wasting and sinking change in the middle of the cancer; thus the growth of its elements is disorderly, and it penetrates its own vessels of supply, starving itself. This is especially true as regards the portal veins, through which indeed cancers of the liver cannot generally be injected; their supply of blood being kept up by the hepatic artery, through which, on the other hand, an injection of their living parts may be usually effected. Another cause of the cupped form of the superficial cancers arises thus, that the centre of the growth commenced when the cancer was small, whilst the margin of the growth grew in its later and always more active time, so that the latter is more voluminous than the former, as one sees in an oyster-shell, where the old part near the hinge is small and depressed.

The cancer, as we have said, is generally secondary. In the only two or three examples we have seen, in which the liver was the only organ affected, the cancer was a rather firm example of carcinoma or scirrhus. In a case which Dr. Pitt considered primary the disease consisted mainly of fibrous tissue, in which were embedded spheroidal cells arranged in tubes and columns. In the other cases the secondary cancer, as usual, has the general character of the primary cancer, but is often softer and more rapid in its growth. The kinds of cancer that occur in the liver are chiefly *carcinoma*, *medullary sarcoma*, and *melano-sarcoma*. To these we may add the *cylinder epithelial* form, already spoken of as secondary to the same in the colon. It is thought by some that a simple fibrous tumour may become cancerous.

*Carcinoma* may be either of medullary softness or of scirrhous hardness; in the former case the cut surface yields, when scraped, a large quantity of characteristic creamy "cancer-juice." It has the usual brainlike character and softness, and does not infiltrate the tissue, but forms large thrusting masses, of which we have spoken. The softening which decay brings in the centre of these cancers is often extreme, so that they may appear like abscesses, and by this softening blood-vessels may be opened and hæmorrhage may occur into the soft space, which thus may become distended into a great blood-cyst. Or smaller hæmorrhages of various dates, with the usual changes in the blood-pigment, may give different shades of brown and purple to the section. When these hæmorrhages occur a similar hæmorrhagic character is usually found in the primary cancer.

Another effect of the invasion of vessels by the cancer is of great interest; thus, approaching a large branch of portal or hepatic vein, it grows into and roughens the channel of the vessel; blood-clot is deposited on this rough spot, and fills the vein. Cancer then grows into the blood-clot, and so a large, soft, carcinomatous plug is found filling the vessel; such great soft cancerous clots have even been found without any implication of the vessels' wall. We have certainly seen such examples. Virchow thinks that these cancerous emboli are not uncommon.

But another source of similar cancerous obstruction of the portal vein consists in *embolism*, as when cancer of the stomach enters and fills the gastric veins. In one remarkable case a continuous cancerous thrombus extended from the stomach cancer, through the gastric and splenic veins, and the whole length of the portal vein, into many of its smaller branches within the liver. This thrombus stuffed the vessels like sausages, but was only here and there attached to their wall.

Besides this extension within the veins, we occasionally meet with very hard cancer taking the place of Glisson's capsule around the portal veins, and also commonly surrounding the gall-bladder. Such growth is, however, generally rather sarcomatous or lympho-sarcomatous than carcinomatous in structure.

*Scirrhus* is often of the infiltrating kind. All varieties of consistence, from the medullary kind just mentioned to an almost cartilaginous hardness, are met with in cancer of the liver. The physical cause of the difference in consistence lies in the proportion which the fibrous network bears to the cellular contents of its interspaces. In scirrhous cancer, the cells are few and soon degenerate, so, indeed, that in the older central part little more than interlaced fibrous tissue may be found, with fatty relics of cells; while in the outer parts, of recent formation, the cells are more numerous; the naked eye sees a reticulated or hyaline appearance.

Sometimes the cancer is seen effused as milky white patches through the liver, and in which the lobules often retain their form. Our Museum shows specimens of this, as well as of others where the cancer is so hard and striated that the section resembles cirrhosis; indeed, it might be said that a cancerous and cirrhotic condition were combined.

*Melanosis.*—Wherever this exists to any extent in the body the liver is liable to be affected. It may be seen in our specimens scattered through the organ in the form of black nodules, or as black lines or patches of infiltration. We speak of melanotic cancer and melanotic sarcoma. The latter would be firmer and formed of spindle-shaped cells.

*Colloid.*—This probably never occurs in the liver as a primary disease, but the organ is sometimes involved when the peritoneum is affected, as in the beautiful specimen already mentioned.

*Epithelioma* as a secondary growth, as already mentioned, has occasionally been found in the liver.

Much attention has been given of late to the mode of formation of cancer of the liver, and we have already spoken of new growths originating in the bile-ducts. There are many good pathologists who believe that the cancer-cells are developed by direct transformation of the hepatic cells. The latter may be seen undergoing proliferation, the nuclei dividing, becoming more numerous, cells formed, the angles being rounded and gradually transformed as Dr. Creighton has shown. We have already seen how spindle-shaped cells and connective tissue are supposed to have the same source. These growths of cells, and fibrous stroma take place in connection with the hepatic artery from which they may be injected.

*Cysts.*—Besides hydatids, we find sometimes upon the surface of the liver small cysts, with very thick walls; sometimes they may arise from *obstructed ducts*, as in the case where there was a chronic induration of a part of the liver from injury, causing obstruction of a duct and the formation of a large cavity filled with inspissated bile; and the same appears to be the cause of the cysts in other preparations.

**Cystic Disease** is seen in some very remarkable specimens associated with a similar cystic disease of the kidney. The portion of the liver which is preserved is full of cysts of all sizes, some as large as a walnut, and others smaller; they are separated by membranous partitions, and on careful examination of the ducts we cannot discover that they are in any way connected with them. Cruveilhier gives drawings of livers containing cysts, which he thinks depend on the dilatation of the ducts. Such may no doubt be sometimes the case, but the present specimen appears as a real cystic disease such as occurs in other organs. In several cases it has been found in connection with a similar disease of the kidney and in a case related by Dr. Hale White with cystic disease of the brain. In a communication made to the Pathological Society, Dr. Pye-Smith showed how the cysts were formed by vacuolation of the liver-cells. Small vacuoles first occurred, and then these ran together until the cell was destroyed and nothing left but a large empty space.

We have other specimens containing very minute cavities or cysts, but whether they be pathological conditions we are not quite satisfied, for we must remember that in very rapid decomposition, a similar appearance may be produced from the development of gas in the tissue; thus a liver we met with not long ago resembled a mass of well-raised dough, and looked very much like our preparation.

**Cavernous or Erectile Tissue.**—We have several specimens on our shelves called blood tumours, supposed in some instances to have resulted from injury, but they are in reality distinct formations or growths of a cavernous tissue. In one specimen we see merely a red tumour, but in another, from which the blood has been washed out, a very beautiful reticulated structure is left. Such formations are mostly met with on the anterior edge of the liver, where we have several times seen them; they are of about the size of a marble, and of a deep red colour. When cut across we see a cellular structure, the spaces or alveoli being filled with blood. Although they are called tumours, yet they do not project from the liver nor compress it; indeed, the affected part is often sunken slightly below the suface, upon which it is seen before section as a dark spot with an angular outline. They have been considered by some to arise in the areolar tissue and to become connected with the blood-vessels afterwards, and Virchow considers that these cavernous patches are essentially tumours with dilated venules, and may be connected with any vessel. The amount of fibrous tissue in them is generally very slight; the network very delicate, and covered with a single layer of squamous epithelial cells of elongated form. They may be injected from the portal vein or from the hepatic artery. In a case of Dr. Payne's the sinuses were attached to and grew from the portal veins. As to their importance, they are chiefly of scientific interest; they were formerly thought to represent an early stage of malignant growth, an idea which was easily reached when such growths were thought to be due to changes in extravasated blood. We have seen them associated with cancer on two occasions, but there was no sign of transformation of the cavernous tumour into cancer; they are not very unfrequent, and usually occur in healthy livers. The only change we have seen in them was a solidification of a part of one, evidently due to coagulation of the blood in it, as it presented all the characters of degenerating blood-clot.

**Leukæmic Liver.**—The liver in leukæmia is found generally very large, and its substance is diseased in a peculiar and characteristic way. Sometimes one can see on section minute grains resembling miliary tubercles, but soft, scattered in immense numbers in the course of the portal venules; generally, however, the distribution of the diseased change is so minute that it is not visible to the naked eye; a paleness and largeness of the lobules only being observed. Microscopic examination reveals collections of crowds of lymphoid cells, mostly following the course of the capillaries; these cells are very like those white cells whose excess in the blood constitutes leukæmia; differing from common white blood-cells, if at all, only in this, that the nuclei are larger and of bolder outline. For a further account of leukæmia see "Spleen."

In the disease known as *lymphadenoma* or Hodgkin's disease, the lymphoid tissue is scattered through the organ in visible masses, usually seen in sections of the portal canal the size of a split pea, and in some exceptional instances as large white masses occupying a large part of the organ. This is usually accompanied by a similar deposit in the spleen. When the deposit is large it has sometimes been called *lymphosarcoma.*

In the disease known as *myxœdema* Dr. Ord believes there is a material resembling mucin penetrating the tissues of the body, and amongst others the liver. He has shown drawings with an intercellular accumulation consisting either of a finely fibrillated or finely granulated matter with numerous large nuclei.

In the skin disease known as *xanthelasma*, the patients have often been observed to be yellow and the liver enlarged. In a fatal case the hepatic duct was completely closed by fibroid thickening.

*Tubercle.*—We have already seen that tubercles appear in many organs under two wholly different conditions correspondingly different in their forms. These are the primary local tuberculous nodule or mass which becomes caseous and softens, and the secondary shower of scattered minute grains familiarly known as miliary tubercle. These tubercles form small greyish or yellowish granules in the tracks of the small portal venules, or hepatic arteries, many usually reach the surface, and upon these may be seen little patches of chronic inflammatory lymph, sometimes making such livers look sprinkled with villosities. In cases of this kind the hepatic disorder forms a small and unimportant share of the whole disease. It is true the liver is usually swollen, but a great part of the swelling is due to congestion caused by the obstructed circulation which the coexisting tubercles in the lung have created. We have already spoken of an indurated liver of a granular kind, and which is often called tubercular, so that some authors do not hesitate to speak of a tubercular cirrhosis. The other form is more rare, and seen as small caseous masses breaking down, forming cavities in the course of the portal canals. They consist of cells and nuclei in a connective tissue, and might be called a kind of tuberculous inflammation.

*Hydatid.*—This is more common in the liver than in any other organ; a fact which tends to bear out the history already given, in which it was shown that the ova of the tapeworm are taken up by a portal vein from the intestinal canal, and are then developed in the liver into round or cystic animals. Hydatids are generally single in the human subject, and so differ from the multiple hydatid seen in the livers of the sheep and other creatures; more rarely, however, two or three of them are found together in the human liver.

The cyst may be of any dimension; it may grow as large as one's head, and contain several pints of fluid; or it may die when young and of very small size. Such dead hydatids are constantly being met with on the post-mortem table, in cases where their presence has never been suspected. The hydatid itself consists of sacs composed of a thin pearly-white translucent membrane, which, when torn, curls back the reverse way to its natural curve. If the animal be large, its wall is of tolerable thickness, and is laminated in structure; but it is so tender that it will scarcely bear its own weight when lifted from its position. Unless some accident has happened to the creature, its wall is tensely full of a fluid like water, of very low specific gravity, seldom reaching more than 1010, and containing a little chloride of sodium; only the slightest degree of opacity is communicated to this fluid by boiling, so that it resembles pretty closely the cerebro-spinal fluid.

The animal generally contains a number of smaller ones of a similar kind, and of all sizes. The large parent sac is lodged within a hollow formed in the liver, but having no connection with it; thus the hydatid drops out when the hepatic *cyst* or containing cavity is opened; the latter is formed in the liver itself in the same way as the wall of an abscess, by a fibrous induration of the hepatic tissue around the hydatid. As the animal grows, so the hepatic tissue gives way, and becomes hardened to form a wall around it. When the hydatid is situated near the surface of the liver and protrudes, the hepatic cyst is protruded also, thus appearing as if it were formed around the hydatid by some independent process of its own, but this is really not so.

The hepatic cyst, under these circumstances, often becomes very hard or bony; that is, earthy matter is deposited in its walls. When found accidentally, hydatids are mostly dead, and then the parent hydatid has shrunken, together with the smaller ones within it; these, instead of floating in the fluid of the parent, are all collapsed and bound tightly together, and as might be imagined under the circumstances, a number of collapsed spheres being pressed together, they encircle one another so as to form many apparently crescentic membranes. In these cases the sac-like bodies have degenerated, and a quantity of fatty or putty-like matter is found amongst them, in which a quantity of concrete bile-pigment is not unfrequently present. The Museum contains a case of Dr. Goodhart's where an immense hard gummatous tumour held an hydatid in its midst.

If the surface of the hydatid membrane be looked at beneath the microscope, it appears to be structureless, but in section it is seen to have a number of concentric linear markings indicating a lamellated structure; sometimes the sac is barren, and when open is found to contain a clear fluid and to have no animalcule-buds attached to it. When this is the case there is most disposition to recovery by simple tapping, such hydatids really representing abortive specimens. More frequently the larger sac, and perhaps the secondary sac also, will have on their inner surface crowds of minute bodies looking like white grains. The inner surface itself is composed of a delicate translucent membrane in which these grains arise. Under the microscope they resolve themselves into clusters of exceedingly small round bodies, each cluster being enclosed in a delicate membrane, and attached by a stalk, which gradually narrows until they separate and become free. The minute round bodies which compose the cluster are found to show the anatomical characters of the head of a tapeworm. That is, they are armed with a close double circlet of well-formed hooks, outside which are four oval suckers at regular intervals a little distance off. The circlet and suckers, with the head which carries them, may be seen protruded in some of the examples from the little sac with which the head is continuous, but more often the head is retracted in an inverted state within the saccular neck, like the top of a glove-finger pushed in. Numerous grains of lime salts mark the surface of this sac. If the hydatid be decayed, the hooklets may, nevertheless, be found, since from their stony nature they are but little perishable. These heads are the young *scolices* of the *Tænia echinococcus*, a small tapeworm of not more than three joints, found usually in dogs. Long before their real nature was known, these heads were recognised by Dr. Bright, as we see them figured, though very vaguely, in the first volume of our 'Hospital Reports.'

We have once met with a case in which an hydatid sac budded from its outer surface, and this process repeated itself so as to form a great chain of hydatid sacs, which extended through the diaphragm, and permeated nearly the whole of the lung without creating any inflammation of its texture. We have already mentioned the occurrence of a similar form of hydatid budding from the outside, in the bones, and elsewhere. Neither of these two examples had any scolices with hooklets present; all stages of the formation, from the budding to the completion of a double cyst, and the repetition of the budding, could be seen; and thus the whole hydatid disease formed a chain of cysts of the kind called multilocular, occupied by the more or less mutually connected hydatids, the connection usually having broken down.

Several German authors have described a minute multilocular hydatid disease, extending in the course of the portal vein through the liver, and producing symptoms of obstruction of the ducts with fatal jaundice, but we have not met with any specimen of this.

The most usual termination of an hydatid is to cease to grow, die, and shrivel up. It may be killed by the entry of bile into its cavity. Or the fibrous encasing cysts of compressed and altered liver tissue may calcify; this, however, is more likely to be a consequence of slowness of growth in the parasite than the cause of its death. The opening of a small duct by the hydatid so as to poison him with bile is good for the host, but it is very unfortunate if a large duct be opened, and the hydatids attempt to pass that way; great pain, jaundice, and death are the almost constant result; hydatids sometimes broach the vessels, and so cause hæmorrhage into their interior. If they continue to grow, their tendency is ultimately to burst, and make their escape. They may be broken by an accidental blow, a very fatal occurrence; or they make their way into the pleuræ, or pericardium, stomach, colon, or into the vena cava, or outwardly through the skin. The risk of either of these occurrences is proportioned pretty closely to the extent of liver surface in contact with these several parts, so that, as would be expected, the right pleura against which through the diaphragm the liver so extensively abuts receives the largest share; commonly the lung becomes adherent, so that the hydatids are discharged through that organ, and may be spat up; this is again a very dangerous event. The most favorable direction is that into the alimentary canal, and many recoveries are recorded of cases wherein hydatids took this course.

*Actinomycosis* is an inflammatory affection set up by a certain fungus, the *actinomyces*, or ray fungus. It was discovered only a few years ago as a cause of disease in the bones and lungs of oxen, and of late has been found to attack man. Some old specimens in museums, styled tubercular, have been found to be the subject of this parasite. It was more especially brought before the profession by Dr. Acland and Mr. Shattock from cases observed in St. Thomas's Hospital. In these the liver was full of abscesses apparently from the breaking down of tubercle, but upon examination found to consist of these peculiar organisms. The latter were described as rosette-shaped masses with a central mycelium from which radiated the conidia or club-shaped processes. These form gland-like bodies resembling mulberries. They finally set up inflammation and abscesses.

# DISEASES OF THE GALL-BLADDER AND DUCTS

**Malformation and Atrophy.**—A congenital obliteration of the bile-ducts is sometimes met with, an example of which may be seen in the Museum. Infants die of jaundice a few weeks after birth, and there is found an obliteration of the bile-ducts or gall-bladder. In adults the gall-bladder is sometimes absent; thus in one case we found the gall-bladder represented by a shrivelled relic of fibrous tissue; death having occurred from painless obstruction of the ductus communis by a large gall-stone. In another remarkable case of extreme obesity the gall-bladder was coated thickly with fat, which had so impeded its function that it contained only a little clear mucus, while the rest of the biliary system was full of bile. Its size varies in many forms of disease; where little food had been taken, or in actual starvation, it is found very full of bile. Where it is much distended an obstruction has existed to the discharge of its contents into the intestine; and such a case occurred here a few years ago, where the gall-bladder held more than a pint of fluid. This condition is called *hydrops vesicæ felleæ;* but it is rather an inflammation than a dropsy. Cases are recorded where, from injury and subsequent fistula, the bladder has discharged itself externally, and bile has been poured out to the amount of eight ounces daily for a period of several months. It is sometimes found sacculated.

**Dilatation.**—We not unfrequently find the gall-bladder of large size where there is no evident obstruction to its duct; the enlargement being evidently chronic, as shown by a deep notch which its pressure has caused in the liver. We have never seen any evil results from such simple dilatation, though on one occasion five ounces of bile were present in such a dilated gall-bladder, without any jaundice. But the occurrence is not without interest in connection with similar dilatations of the stomach, urinary bladder, heart, ureters, &c., without evident mechanical cause. It appears that any hollow organ may slowly yield through inability to expel its contents, but it is always more satisfactory to find a tangible obstruction, which should be carefully searched for.

The most common cause of dilatation of the gall-bladder and biliary ducts is a closure of the hepatic or common duct, by cancer or by a gall-stone, in fatal cases of jaundice. Behind the obstruction the ducts become enormously widened, so that they may be seen projecting from the surface of the liver; and they appear when opened as large as one's fingers, very frequently pouring out fluid to the amount of half a pint. When the dilatation is extreme this fluid is not bile, for the secretion at length becomes so impeded (the bile which previously existed having again been taken up into the blood), that nothing is left in the ducts but a thin mucus often perfectly colourless.

Now, when we find the body most deeply jaundiced, and the blood-serum dark orange-yellow with bile-pigment, it is surprising to discover that the gall-ducts, which are supposed to be the source of the bile furnished to the blood, are themselves devoid of bile and contain a colourless fluid. This indeed proves that the gall-ducts are not the source of the bile, and that the bile in the blood in these obstruction cases is absorbed from the liver tissue, if it be absorbed at all. The question is thus raised whether it is not by suppression of secretion that the jaundice is caused as it is supposed in the unobstructive jaundice of yellow atrophy, fevers, &c. The clear white slightly viscid fluid present in the ducts under the circumstances is a thin mucus secreted by the walls of the ducts in which numerous mucous glands are embedded. There is commonly no albumen in the fluid, and even by the nitric-acid test no colouring matter of bile can be discovered. We shall see in our specimen how enormously these ducts become dilated; sometimes small pouches are formed in them; the liver is then of a dark green colour, and it was stated by Dr. Thomas Williams some years ago, in our 'Hospital Reports,' that the secreting cells became destroyed; and no doubt there is an important truth in this statement, although the liver-cells are not altogether broken down, but rather shrivelled and defaced and pigmented, and only here and there quite disintegrated. In some cases, however, the destruction of cells is very marked; the microscopic condition approaching that found in yellow atrophy. It is here important to notice that the fatal termination of such cases of obstructive jaundice not unfrequently takes the form of apoplectic coma from the blood being poisoned by bile, a condition to which the term cholæmia has been applied.

**Acute Inflammation** of the ducts has sometimes arisen from injury, and has occurred in cases of general suppuration of the liver which has involved these parts.

We have met with a few cases of acute idiopathic inflammation, the gall-bladder being thick walled and reddened, and containing mucus. Sometimes a layer of mucus adheres to the wall whilst the bile occupies the centre. As an idiopathic affection, this is extremely rare. We have found it in some cases of acute yellow atrophy of the liver, and on other occasions it has been associated with gall-stones, probably as a result of irritation by these concretions. Such inflammations of the gall-bladder with formation of pus in the wall, or great infiltration of it with lymph, or ulceration of it, have been met with in fevers and in cholera.

Catarrh of the bile-ducts is a term which is frequently employed at the bedside to explain the occurrence of simple painless jaundice. As these cases all recover when uncomplicated, they may be said to have no morbid anatomy; so that it is only as a matter of speculation that inflammatory mucus or turgescence of the mucous membrane is assumed to exist.

We can easily prove by experiment that no pressure of the gall-bladder through the parietes could exert any important pressure upon the ducts; so that no good could be expected from this proposed plan of treatment; but the gall-bladder, if thin or ulcerated, might be burst, an accident which has occurred during such attempts.

**Chronic Inflammation and Ulceration.**—These states are nearly always due to gall-stones, or at least accompany them. But a few cases of simple stricture are on record. The changes which gall-stones produce in the gall-bladder and on parts around make one of the most intricate and important chapters in pathology. The simplest condition we have observed, as a result of gall-stone, is a widening and slight thickening of the whole length of the duct from the gall-bladder to the duodenum; this wide duct having several gall-stones in it. We have twice known this occur when relatives of the person have denied the presence of pain or other biliary symptoms during life. Indeed, gall-stones are not unfrequently latent; this is, however, usually not because they pass without symptoms, but because they do not pass out of the gall-bladder. It is not very uncommon to find a large gall-stone firmly impacted in the neck of the gall-bladder, entirely closing it. The bile in the gall-bladder is then absorbed, and replaced by a thin mucus; and the walls of the gall-bladder are usually more or less thickened. Sometimes gall-stones produce severe inflammation of the gall-bladder, causing it to adhere to parts around, usually the duodenum, and by ulceration making a perforation by which the gall-stones are discharged. In this way very large gall-stones may be got rid of, but many risks of fatal danger beset this kind of relief. If the stone is large it may block up the small intestine and kill by obstruction; this end is relatively not very unfrequent; or the ulceration may set up pyæmic abscess of the liver, or sometimes suppuration extends by continuity along the ducts, in the tract of the portal vein. Sometimes the obstructed gall-bladder suppurates and forms a large abscess. It occasionally happens that the gall-stones are discharged through the abdominal wall; this may occur without any previous jaundice, and so the case be mistaken for carbuncle, &c., the real nature of the case being further obscured by the course of a sinus for some distance through the abdominal wall so as not to point over the gall-bladder.

More rarely the gall-bladder opens into the colon. In two such examples we recently met with, the gall-bladder was the seat of extensive cancer, and this is usually the case when the colon is opened by it.

In yet other examples the gall-bladder opens, commonly by rupture, into the peritoneal cavity, and, again, in very rare instances gall-stones have been passed in the urine. It is almost doubtful whether the gall-bladder ever communicates directly with the stomach in this manner, though the opening may be close to the pylorus instead of about the second flexure of the duodenum

Gall-stones, however, are occasionally, but very rarely, ejected by vomiting.

**Morbid Growths.**—Cancer in these parts is not very unfrequent. We have met with several cases where a small knot of cancer has grown in the bile-duct wall, and proved fatal by a very chronic painless jaundice. Once the hepatic duct alone was affected by a very remarkable growth, the substance of the formation resembling the local hypertrophy of gland-tissue in the mucous membrane of the mouth we sometimes meet with. In this case, as in other very prolonged obstructed cases, *xanthelasma* was present. A more common seat of small tumours obstructing the duct is about the ductus communis, near the head of the pancreas. We have twice known syphilitic gumma form in this region, and cause jaundice by its pressure on the duct; once it softened and burst into the duodenum, and so the jaundice was relieved.

In other examples the new growth extends along the course of the portal vein, appearing as a whitish thickening of Glisson's capsule, and perhaps extending to and involving the gall-bladder. Some instances of this offer a not uncommon difficulty in distinguishing between inflammatory thickening and tumour, the substance following the course of the ducts, and being of an almost purely fibrous structure, with scarcely any cell-element. One specimen we have seen was composed of lymph-gland-like substance, and the disease was associated with a similar change in the wall of the duodenum.

We recently met with a curious example of disease. The common duct in a child of four years was dilated to form a cyst as large as the child's head. In the wall of this cyst were crowds of pendulous growths of myxomatous structure, in which were embedded numbers of non-striated muscle-fibres. The gall-bladder itself is sometimes primarily affected with cancer, which may enlarge it enormously; it then usually contains gall-stones, and it may, as we have said, open into the colon and discharge these. A consideration of the history of such cases leads sometimes to a belief that the cancer follows upon the gall-stones as a result of irritation of the gall-bladder through their presence. Twice we have seen this cancer take the villous form, the cavity being enormously distended, and occupied by crowded dendritic growths. The liver may be invaded by contiguity of such cancers, or secondary growths may arise in it.

*Ossification, fatty degeneration, &c.*—In cases of xanthelasma from chronic obstructive jaundice, where the ducts were greatly dilated, we have seen the lining of their walls in a state of fatty degeneration similar to that which produces the patches in the skin. Occasionally we find a calcareous degeneration of the gall-bladder and ducts, the inner surface of these being covered with white deposits of carbonate of lime; this seldom reaches to any extent, or approaches to what is seen in the liver of the sheep or of the ox. Thus, in a specimen from the latter animal we see the ducts forming rigid long tubes, and within them are flukes (*Distoma hepaticum*); but whether the parasites are the cause of the disease we cannot say. In speaking of parasites it has been stated that the *Ascaris lumbricoides* has occasionally penetrated the gall-ducts; and the proof that the worm had reached there before death was the fact of its having caused an abscess around the part where it was located.

**Gall-stones.**—The usual seat of these is the gall-bladder, as we should expect, or else some point in the course from the gall-bladder down the ducts to the intestine; but now and then biliary concretions are found in the hepatic ducts within the liver. They are then generally quite minute, constituting what has been called biliary gravel; but sometimes, as in one of our specimens, we see large calculi in the ducts of the liver itself. The cause of the production of biliary calculi has never yet been clearly ascertained; they are constantly met with when the liver is quite healthy, and this we also know from the fact of many persons being in perfect health after passing them. They may, however, coexist with cancer, cirrhosis, and other diseases. The tendency to their production increases with age. They consist of elements of the bile, and we can easily imagine how these might form concretions from some change or decomposition of that fluid, whether this occurred spontaneously or from disease of the coats of the gall-bladder. The constituents of gall-stones are cholesterine, bile-pigment, and earthy matter; the last two combine in a definite compound called pigment lime. Some calculi are formed of pure cholesterine, and then constitute very beautiful translucent crystalline bodies. In other calculi this substance may be seen as sparkling pearly plates, or, if not visible, may be procured by boiling a piece of the stone in alcohol, and pouring the solution on a slide, when the crystals readily form; they are easily recognised under the microscope as rhomboid plates, lying one over the other, and with one angle often deficient, the two perfect angles at either end being 100° and 80°.

Most gall-stones, if of any size, are composed of earthy matter, pigment, and cholesterine; and if one be split across it is seen of a brownish colour, and formed often of concentric layers, with pearly plates glittering amongst them; the centre is often black, as if of pure pigment, and from this some radiating lines may be seen. A combination of all these substances is the most common, but sometimes the calculi are black, and of rough irregular surface, as if composed of pure pigmentary deposits; at other times of a white colour. Sometimes we see the gall-bladder full, and the calculi polygonal, from the attrition of one against the other; or rather, we believe, in many instances, simply from their compression against one another during their increase. More rarely they are found in the ducts of the liver, as we have already said, but if so, they may put on an irregular branching shape. As regards the cause of their formation, we know nothing positively, but we think it can be easily seen how very readily they might be produced. We have said that the cholesterine, pigment, or biliverdin, which constitute calculi, are also ingredients of the bile, and therefore it can be readily perceived how, if once a nucleus existed, a precipitation and concretion would result in the same way as the urinary ingredients form a calculus around any foreign body in the bladder; and if there should be no such cause, we can see how a change in the constitution of the bile might lead to a precipitate, for besides the substances already mentioned which exist in bile, there is another, its principal constituent, a resinous substance, or the *bilin* of Berzelius, which is now known to be a compound salt, called the cholate of soda (glycocholate and taurocholate); if some of this salt be added to a gall-stone it will cause its solution, whence it can easily be understood how any cause operating, either in the liver or the walls of the gall-bladder, to alter the character of the secretion, and destroy or lessen the amount of this peculiar principle, would tend to the precipitation of the other ingredients; but whether such method is in operation for the production of gall-stones we do not know.

Dr. Ord, from experiments made with cholesterine and pigment, concluded that a calculus is of slow formation; that spherules first are formed and then crystallization slowly proceeds. The interior is usually radiating and crystalline; around this are seen concentric layers of varying colour with an outer layer having no radiation.

# DISEASES OF THE PANCREAS

Diseases of the pancreas are not very common; we mean those diseases which are connected with great structural changes; that is, such as are so frequently met with in the lungs and elsewhere. The pancreas shares this immunity with the salivary glands, to which it is allied very closely in structure and function. It is very probable, however, that more minute alterations in the secreting structure may be constantly occurring during life, and that these are productive of symptoms in the same way as variations in the secreting tissues of the stomach, &c.

**Inflammation.**—*Acute.*—Although it is probable that changes may occur in the secreting tissue more or less allied to those called inflammatory in other organs, yet the more marked effects of inflammation, such as suppuration, &c., are not often seen. We have met with abscess on more than one occasion around the pancreas, and involving it, but it was probably only secondary; thus, we have met with suppuration of the pancreas in the later stages of typhoid fever. The other salivary glands likewise occasionally suppurate after fevers, pus forming about the gland as well as within it. We have met with it in connection with thrombosis and with calculus.

We have also specimens on our shelves which are described as *gangrene* of this organ. As we may sometimes find the pancreas involved in any suppuration which may be going on near it, so we may find the remains of inflammatory products within it. We have more than once found the pancreas occupied with white amorphous matter, the remains of pus or other unorganisable exudation; in one case this was connected with a perforating ulcer of the duodenum which was undergoing cure.

*Chronic inflammation and induration.*—Chronic inflammatory changes of the pancreas are described by some authors to which the name cirrhosis has been given. The anatomical evidence of such occurrence is not very satisfactory. But the pancreas is found to vary frequently in size and hardness; the clinical importance of such changes has also yet to be proved. It may also be involved in suppurative and gangrenous processes.

The most important effect ascribed to this induration of the pancreas used to be a compression of the common bile-duct when it joins the pancreatic duct, and the consequent production of prolonged deep jaundice and wasting. We have occasionally seen cases in which the pancreas shared in a chronic thickening of the tissues about the bile-ducts, and generally of Glisson's capsule. The jaundice which accompanied these cases was due to the constriction of the biliary canals by the chronic fibrous formation around them, and not to the pressure of the indurated pancreas. The impeded function of this organ, however, doubtless aggravates the symptoms.

The pancreas is apt to be involved in ulceration of the stomach (this organ, we know, is placed behind the lesser curvature of the stomach, as the latter curves round the spigelian lobe), and thus it sometimes forms the floor of a large gastric ulcer, while the ulcer in spreading opens either the splenic or one of the pancreatic arteries, causing speedy death by hæmorrhage.

*Atrophy.*—Dr. Lancereaux and some others have published cases where atrophy of the pancreas was found in "Diabetes." We have ourselves seen several examples of it so that the subject is open for further investigation. It has been found as simple wasting or fatty degeneration.

**Morbid Growths.**—We may here mention the occasional occurrence of an accessory pancreas; one or more of these may be found forming gradations between the pancreas and Brunner's glands, which resemble each other very closely in structure.

*Cancer.*—No part of the body is free from a liability to cancer. This proposition is especially true if, by cancer, we mean tumour which is malignant in the sense of causing death—a latitude of signification too frequently allowed to this most unruly word. We use the term cancer only for malignant, that is, auto-infectious, tumours; but as we have seen elsewhere, tumours in these vital parts, although of an infectious nature, yet may destroy life before secondary growths have had time to arise; hence it is only by careful study of the structure of these tumours, comparing their anatomy with the different kinds of tumour found elsewhere, that we can decide upon those proper characters which their premature mortality nipped in the bud.

No doubt it is very wrong to speak of the chronic inflammatory indurations we recently referred to as *scirrhous*, and to confuse them with malignant disease; but other tumours occur which have all the characters of true carcinoma, ranging in feature from the soft and encephaloid to the hard contractile scirrhous form. Particular interest attaches to those cases in which a small knot of true scirrhus arises in or near the head of the pancreas, and drags upon, compresses, or otherwise closes the bile-duct, and perhaps the pancreatic duct also, leading to tedious but generally painless jaundice and wasting. The pancreas, like the other salivary glands, has many lymphatic glands around it; and again, its substance is not enclosed in a capsule defined by a sheath of peritoneum, so that, as we have seen to be the case with the other salivary glands, it is often difficult to decide whether a tumour partly involving the pancreas did or did not originate in its proper substance. The pancreas is often involved in cancerous or malignant disease; but it is often impossible to say whether the disease began in the pancreas or in the glands about its head. It is certain that these glands do become involved in cancer whilst the pancreas escapes; and this has induced in some the belief that the pancreas nearly always gets its disease by extension from the glands. Nevertheless, we have met with a few cases where the disease was limited to the pancreas, but differing from the primary cancer of the lymphatic glands, which is almost always *lymphoma* in being true *carcinoma*. Dr. N. Moore describes a primary cancer originating in the epithelium of the acini, and also an adenoma-sarcoma.

We have known the pancreas to have been affected with a *colloid* form of cancer; thus, in one of our examples nearly the whole of it was involved, the only other disease in the body being a similar affection of the omentum. Death was slow and accompanied by excessive emaciation.

*Tubercle.*—We have never seen an instance of tubercle in the pancreas; both it and the salivary glands appear to enjoy almost complete immunity from this form of disease. Dr. Barlow, however, has lately reported the case of a child aged two years, the subject of general tuberculosis, and in the head of whose pancreas, when cut through, were a dozen white nodules the size of pins' heads. They resembled exactly the tubercles in the kidney. Occasionally the remains of chronic abscess in the form of more or less cheesy dried pus give an appearance such as might be called scrofulous. We have before mentioned a case of syphilitic deposit in the cellular tissue near the head of the pancreas. We have never seen that kind of disease within the organ; its occurrence there must be extremely unusual. We know of but one recorded instance.

**Obstruction and Dilatation of Ducts.**—We have already mentioned that such conditions are very common. In cases of constriction of the common bile-duct in jaundice the pancreatic duct may also be involved, and then, just as the hepatic ducts are dilated behind the stricture, so is the pancreatic duct widened, as seen in our specimens.

Occasionally *cysts* are formed in the pancreas, as our specimens show; one we met with not long ago was as large as an egg, and contained a white, viscid fluid, corresponding to the pancreatic juice. This has been called pancreatic ranula. We have seen a pancreas with numerous small cysts associated with cystic disease of the kidney and liver.

**Calculi.**—As these may form in the salivary glands, so they may sometimes in the pancreas; they are generally small and multiple. Those analysed by Dr. Golding Bird, consisted of oxalate of lime, phosphate of lime, and phosphate of magnesia. Those that form in the salivary glands generally contain much carbonate of lime, and effervesce freely on the addition of acids.

**Injury.**—The sheltered position which the pancreas occupies places it almost out of reach, except by gunshot or penetrating wounds; but in one example, which was taken from the body of an adult who had been run over in the street, the pancreas was so crushed in its middle as to be divided into two parts. The injury affected the part opposite the spinal column, whose prominence no doubt afforded the counter-pressure in the crushing action. There was but little other sign of injury to the abdominal viscera.

# DISEASES OF THE SPLEEN

**Malposition.**—The spleen is occasionally found in large umbilical herniæ. It is also very rarely found in the thorax through congenital absence of the diaphragm, or through rupture of the diaphragm from injury.

Notwithstanding the looseness and comparative slightness of its connection with the diaphragm, it is not often subject to accidental displacement. This is, no doubt, due to the suction which the elastic lung exerts through the diaphragm upon it. Nevertheless we have once found a spleen of twenty-four ounces weight entirely dislocated, and lying in the pelvis forming a tumour, which might easily have been mistaken for ovarian.

Some cases of the kind have been collected by Küchenmeister, under the name of "The Wandering Spleen." Such dislocation of the spleen must be distinguished from the extension downwards of a large spleen, which still occupies its natural site. When the organ attains great size it may reach and lie in the pelvis extending across this, even to its right side, while it still continues to occupy its normal position by its upper part. Under these circumstances it should be observed that the intestines never lie in front of the spleen. This will often distinguish splenic from renal tumours; while it should also be noted that tumour of the left kidney occupies a more posterior position in the left loin than a splenic tumour. In moderate enlargements of the spleen, such as those caused by fever, the organ is commonly found by percussion to extend itself upwards as well as downwards.

**Malformation.**—It is not uncommon to find the spleen malformed; thus, if we look on our Museum shelves, we find some fissured, some lobulated, and others multiple, that is, made up of, or more frequently supplemented by, a dozen or more small organs. These are merely connected to one another by blood-vessels.

**Congestion.**—No organ is so variable in size as the spleen; indeed, it varies so much that the weight of it in the normal anatomy is very vaguely stated at from four to ten ounces. It is much smaller in elderly than in young people; indeed, it may waste away in old age to a pale relic of not more than two or three drachms weight. The substance of the spleen is most intimately related to the blood which it contains. Some anatomists have described the arteries as opening into spongy splenic tissue, and the veins as emerging from this. Others suppose the pulp to be within the blood-vessels; and yet others, including the chief authorities, agree with Kölliker in the belief that a part of the blood traverses the pulp on its way from artery to vein, just as the lymph permeates its glands, while another part of the blood passes through capillaries in the ordinary way. We give these details because they must be understood before one is at all prepared for the exceedingly variable size and consistence of the spleen. The spleen, indeed, is a most sensitive index of blood changes, and is able to swell very quickly to a large size, as well as equally quickly to return to its normal limits. The mechanism of these changes is not fully understood, but when we apprehend the anatomy of the spleen, and perceive that it is a greatly expansible cellulosity into which the blood has direct and free access by the arteries, we can very easily anticipate that any hepatic obstruction, nay, that any obstruction which should render the flow of blood in the body more difficult, would cause swelling of an organ so ready to yield under blood pressure. The speedy variations of size which the spleen undergoes are rendered possible by the elastic coat of the organ, which favours its return from the distended to the normal condition. The presence of this coat shows a provision for recovery from distension, and, as in the case of the lung, infers that the organ is intended to undergo such changes. In some animals the recovery of the normal size of the organ is not left to its elasticity, but muscular fibres are introduced into the trabeculæ, and then a more active contraction of the spleen is provided. Professor Roy's researches show that the organ undergoes a regular rhythmical contraction.

In ague the spleen is large, and the shrunken, bloodless state of the surface of the body in the cold stage of the ague fit is, no doubt, partially due to the retention of blood in the spleen. Whether the retirement of the blood from the general surface causes the congestion and enlargement of the spleen is more doubtful, and this doubt must be allowed to rest on all explanations of great enlargement of the spleen, which suppose it to be due to mere mechanical obstruction to the exit of its blood.

Moderate swelling, no doubt, occurs from such obstruction, and when the obstruction is complete, as in thrombosis of the portal vein, the size of the organ may be very great, say twenty-four ounces or more. But in ordinary cases of cirrhosis of the liver, although the spleen is often large, weighing generally about ten ounces, yet it may be small; thus frequently we have found it only five ounces in weight, and once it weighed only two and a half ounces in a case where the liver was covered with a thick capsule, and reduced to thirty-two ounces, its atrophy causing fatal hepatic ascites. Again, as we shall presently more fully show, the spleen in obstructive heart disease is usually small. These facts prove that mechanical congestion of the spleen is not the chief cause of its hypertrophy, and we shall find reason to believe that all considerable hypertrophies of the spleen are due to diseases, either of its own proper texture, or of the blood.

The colour of the spleen varies a good deal, corresponding to the several shades of venous blood; relieved, of course, by the presence of the pale-coloured trabeculæ, and other white matters intermingled with the blood colour. As a general rule, the hue is not a consideration of much importance, except in the case of melanæmia, which we shall presently describe.

The colour of a section is always comparatively dark when fresh made; but exposure to the air causes oxidation of the blood, and thus from a dark venous tint, the cut surface changes to a light colour, approaching that of arterial blood.

**Hypertrophy.**—Mere accumulation of blood in the veins of the spleen will not account for most cases of its enlargement; the spleen not only enlarges but frequently also keeps its proper consistence. By hypertrophy of the spleen we mean a condition in which its tissue is simply increased with no alteration of its structure. The spleen is very remarkable for the large size it attains to, while still showing its usual characters in point of consistence, texture, colour, &c. Hypertrophied spleens are sometimes softer or harder than the average; these variations of consistence they, however, share with the normal-sized spleen, which is very liable to alterations of consistence. We have remarked upon the very rapid increase of size which the spleen shows in agues. It is easy to suppose that if the enlargement from such causes were long continued the tissue might lose its elasticity and the organ would retain permanently its increase of size; this is a probable explanation of some forms of hypertrophy of the spleen; but while accepting such simple explanations we must notice what we have already mentioned, namely, that in heart disease, even when the liver is in a state of advanced nutmeg change from chronic engorgement, the spleen is often even smaller than natural; and the same is true also frequently in cirrhosis of the liver, for the spleen is not constantly enlarged in cases of cirrhosis, though it generally is so. But on mechanical grounds the spleen would be expected to be of great size in these diseases. Nevertheless, such facts must not induce us to deny that obstruction of the portal circulation will cause great increase of the size of the spleen, for in those not very unfrequent cases in which the blood in the portal vein coagulates ante mortem, the spleen is almost invariably very large.

The more common cases of considerable hypertrophy of the spleen are, then, not due to mechanical causes. If we should point to any class of affections that specially produce such hypertrophy, it would be to those chronic febrile states which directly implicate the blood; especially we would mention ulcerative endocarditis. In these cases the spleen is large though soft and pulpy, and may weigh twenty-five ounces or more.

On the other hand, the spleen is usually quite small, as well as hard and dark coloured, in cases of chronic cardiac obstruction due to narrowing of the orifices or dilatation of the cavities of the heart;

that is, in cases where *ischœmia* or obstruction to the course of blood is present, without poisoning of its current with inflammatory products. The spleen under these circumstances will generally not weigh more than from four to eight ounces.

In syphilis, again, we often meet with hypertrophic enlargement of the spleen. We do not now refer to the enlargement brought about by lardaceous change. In syphilitic bodies quite free from lardaceous disease, the spleen is often of large size : thus, in eight such syphilitic subjects, the average weight of the spleen was nineteen ounces; no cirrhosis or lardaceous disease being present in them.

Prolonged exposure to tropical miasm is another cause of hypertrophy of the spleen ; the enlargement results even when there have been but slight manifestations of intermittent fever ; and this fact again throws doubt on the simple explanation of the hypertrophy as due to mechanical engorgement, and indicates that there is some special disorder of the relation between spleen and blood in these fevers. In typhoid it is usually enlarged and soft.

We must not say that all chronic febrile states cause enlargement of the spleen; thus phthisis does not do so, neither do the fevers that accompany chronic surgical suppuration, unless the lardaceous change intervene. Indeed, we think that when the spleen is greatly enlarged in any fever, this fact has a signification beyond that of the pyrexia or hotness, and indicates that there is a special disorder of the blood in that fever.

But besides these cases there are others in which the spleen is enlarged to a great size, and this enlargement is the chief, if not the only disease that can be discovered. Thus, Mr. Spencer Wells removed a spleen weighing 6 lbs., this being the only disease discoverable in the body, and the blood not being leukæmic; and Mr. Squire met with a spleen weighing 13 lbs., while the blood did not show a positive excess of white corpuscles. We have met with similar cases of simple hypertrophy of the spleen without leukæmia both in the living and in dead subjects. They are generally, but not always, at least during life, associated with anæmia, and so, indeed, as a rule, are all long-standing diseases of the spleen. These cases are the only ones that can be properly considered as pure examples of hypertrophy of the spleen ; the cause of the enlargement is unknown, and the accompanying clinical phenomena need further investigation.

We are not now speaking of Hodgkin's disease, or anæmia lymphatica; for in the cases at present under consideration the splenic tissue is normal throughout; whereas in Hodgkin's disease isolated patches of peculiar deposit are constantly present in the spleen. The spleen has sometimes been found enlarged in exophthalmic goitre, but in the cases we have seen it was of natural size ; once it was four and a half ounces, or less than the usual weight.

**Leukæmia.**—In leukæmia the spleen reaches an enormous size. Thus one which Mr. Bryant removed weighed 168 ounces, and this is not an extreme weight for the spleen in leukæmia. The tissue of the organ is generally not discoverably diseased, in spite of the great increase of size; indeed, the liver often shows much more of morbid change than the spleen in leukæmia. The liver in the above-mentioned case weighed 138 ounces. The tissue of the spleen in this disease varies somewhat in consistence, like ordinary spleens. Thus it may be rather flabby, or it may be firm and resistant. Its capsule is not generally inflamed or otherwise altered, so that any adhesions or thickenings of it must be regarded as accidental. The surface may present a minutely mottled appearance from fine white lines and dots forming a network in it. The same mottled appearance may be seen on the section, the natural deep purple colour of the splenic pulp being the prevalent colour, but this being mingled with the pale lines, which are sections of the trabeculæ and vessels of the spleen, and with the pale Malpighian corpuscles, which are often somewhat enlarged; in short, the usual appearance of the spleen, but coarser in its markings. The only microscopic peculiarity we have been able to discover is the filling of the small vessels with white blood-cells; but this appearance is of course universal in the tissues. We have several times met, in leukæmia, with *embolic infarctions* in the form of pale, circumscribed, wedge-shaped patches, with a deep zone of congestion round them; both the spleens removed by Mr. Bryant showed these infarctions, although the general health appeared good when the operation was undertaken. In one of these cases there were similar infarctions in the lung, having much the appearance of pneumonic patches found in pyæmia, except that they showed no signs of breaking down.

The blood in leukæmia contains a very large quantity of white corpuscles, so large a quantity indeed that its appearance is much changed, and it has a creamy purplish look, which is very characteristic. This whitishness of the colour is well denoted by the name *leukæmia*, while lesser degrees of increase of the white cells, such as exist in some fevers, &c., receive the name of *leucocytosis.* If leukæmic blood be kept standing some time, the red corpuscles will sink, forming a dark lower layer, above which the white corpuscles float in the serum as a pus-like upper layer. The white cells in leukæmia appear to us to be of larger size, and to have bolder nuclei than ordinary healthy white cells. It is said that in leukæmia from disease of the lymphatic glands, the cells are smaller; but we have no experience of a purely lymphatic leukæmia. This changed state of the blood has a curious effect on the microscopic appearance of the organs. The capillary vessels are found stuffed with the white blood-cells, so that these often serve to mark out the course of the minute blood-vessels in a very useful and interesting manner. We have already described some of the effects of this state of the blood on the liver, where the white cells leave the vessels, and form collections along their course. The same occurrence takes place on a smaller scale in other organs, especially the kidneys, the wall of the intestine, and the heart. In these organs, pale, roundish patches are found, which are very conspicuous to the naked eye ; but when the microscope is used the effect is disappointing ; one only sees that some of the white corpuscles have escaped from the blood into the tissue. It is better not to call these lymphoid formations leukæmic tumours, as it would bring them at once into the category of Hodgkin's disease with which they are not yet proved to be identical, although closely allied. The relation of the altered blood to the affected organs is by no means yet agreed upon. The blood is full of white cells and at the same time many of the organs are crowded with them. It is pretty well agreed that the most marked case of leukæmia is that where the spleen is hypertrophied and there is a pretty general belief that the enlargement is the cause of the condition of blood. In those cases where the spleen is not much enlarged, but the glands, liver, and other organs are increased in size, it is a question how far the glands may have given rise to the leukæmia. Of late, too, it has been conjectured that the bone marrow may have participated in the formation of white cells, since in these cases the marrow has been found of greyish-red colour and composed of a mass of cells or lymph tissue.

The general opinion seems to be that the spleen, lymphatic glands, and bone marrow are instrumental in the production of the excess of white cells, and that the other organs are merely infiltrated with them. Of this, however, there is no proof, and it may be equally true that medulla of the bone may be merely one of those situations where the cells accumulate.

**Atrophy.**—Besides finding the spleen immensely enlarged, we may meet with it equally surprisingly shrunken ; it may be reduced to an exceedingly small size. It is said to have been found weighing only one drachm, and many cases are recorded of its having only the size of a walnut, &c. Under these circumstances the usual varieties of consistence are met with. We do not know that any symptoms attend on this atrophy of the spleen ; it occurs especially in old persons. Considerable wasting of the spleen is natural in age.

**Softening.**—We have several times alluded to the variations of consistence of the spleen associated with its variations of size, but it will be needful to direct special attention to the extreme softening of this organ which is apt to occur in febrile states. This softening is generally, but not invariably, accompanied by enlargement. The softening may be extreme, and may reach such a pitch that the splenic pulp is almost diffluent, and the whole organ like a bag of thick purplish creamy liquid. In this condition it may rupture during life. The microscopic examination of such spleens is very difficult ; nothing is discoverable that can give an explanation of the change ; it is the pulp that is softened and not the trabeculæ, so that a stream of water easily washes the pulp out of the trabeculæ, and thus these spleens serve well to display the trabecular structure. We know no ground for the statement of Rokitansky and others, that the spleen in fevers contains a peculiar deposit, the so-called typhoid deposit. It is true that there may be found an excess of white corpuscles, but then this is true in many other febrile diseases.

**Melanic Spleen.**—*Melanœmia.*—As a result of intermittent fever, pigment is found in the spleen and other organs, especially in the liver, brain, and kidney, as well as also in the blood. There are reasons to think that the spleen is the source of the pigment, or at least the chief source. The reasons are chiefly these: First, the spleen is known frequently to contain, under ordinary circumstances, cells which hold blood-corpuscles in their interior in a more or less defaced condition, and also to contain pigment grains and flecks in its pulp. Secondly, the spleen is commonly severely implicated in intermittent fevers, which cause melanæmia; nevertheless, we cannot conclude that the spleen is the sole source of the pigment, since in a case observed by Frerichs, the pigment was found in the liver, and not in the spleen.

There is little to describe in these spleens, except the change of colour, which becomes either greyish black, or bluish black, or dark brown. The consistence varies as usual in all splenic diseases, being softer in the early stages, and denser in the latter; but this rule is subject to the accidental cause of the patient's death. There is generally some enlargement of the organ.

Pigment is found both free and enclosed in cellular bodies of various forms, of which some resemble the spindle-shaped corpuscles that line the splenic vein, and others are of a more rounded figure. The free pigment also varies much in its physical condition. In well-marked examples of the disease, the blood is found to contain pigment in much the same forms. The discovery of the pigment in the blood, which first threw light on the nature of melanæmia, is due to H. Meckel. It is believed that the pigment formed by the spleen passes from this into the portal blood, in which it reaches the liver, where its coarser particles lodge in the outer zones of the lobules; the finer particles pass through the liver, and are found in various parts of the body, not universally. Thus, they do not discolour the muscles nor the skin; at least, not constantly or characteristically, though a sallow hue of the complexion is often present; but the brain and kidneys show the black colour very frequently. Thus the process may be described as microscopic embolism of the affected organs, with pigment produced in the spleen. In many cases observed in America, the pigmentation was found to be limited to the portal blood.

In this country little attention has been given to the disease. It is, indeed, rarely met with here, since the conditions of severe and prolonged ague, to which melanæmia owes its origin, have been almost entirely dispelled by the drainage of marshy places in our island. We have met with but two examples of this disease, and can add nothing to the already very full descriptions of it; but great interest attaches to the drawing of a brain from Dr. Bright's 'Medical Reports,' which undoubtedly represents this affection. The case is described as that of J. C—, an old man, who, accompanied by his wife, set out from Horncastle, in Lincolnshire (an aguish locality), in August, 1829, for London; and having walked all the way, fell ill immediately on his arrival, and was brought to the hospital in a dying state, apparently with fever. On post-mortem examination the brain was found as represented, having its superficial cineritious structure of the colour of black-lead, but the section showed the medullary part unaffected, except the puncta vasculosa, which were found of the same blackish colour, and the vessels were filled with dark blood. The spleen was also of a dark hue. The wife's brain and other organs were also dark, though less so than those of her husband.

Although the anatomical facts concerning melanæmia are well established, and their interest is very great, yet there are conflicting opinions as to the importance of the disease.

**Enlargement of the Splenic Corpuscles.**—We find great varieties in the size of these bodies in different cases, but it is doubtful whether they have any pathological significance. Experiments have shown that these corpuscles, or "Malpighian bodies," vary with the stage of digestion, and this will sufficiently account for the different sizes they present to the eye, sometimes being large and at others invisible. One point of interest respecting them is their very great size in some fatal cases of purpura which occurred a few years ago, but the observation has not been confirmed.

**Injury.**—This is not uncommon from blows on the abdomen; if the splenic vein be opened the blood may drain from the vena portæ, when the first thing noticeable is the black pitchy character of blood effused. The spleen may be burst open from unequal pressure on its surface, or may be transfixed through the diaphragm by fractured ribs. Injuries of the spleen are by no means necessarily fatal, for cicatrices may sometimes be found on this organ; and in cases of accident from falls, &c., which are not immediately fatal, and in which the spleen has been lacerated, the various stages of repair may be seen. We have one with lymph on the surface a week after the injury, and in another case five ounces of laminated clot, in process of absorption, are seen lying on the surface of the spleen. The death occurred eighteen days after the accident, and was caused by abscess of the brain.

We have already alluded to reported instances of spontaneous laceration of the stomach and liver from violent efforts during vomiting; but there can be no doubt that the spleen may give way from undue distension, such as occurs in intermittent fever; there are many authenticated cases of the occurrence. That which, however, is perhaps more frequent in India and other hot climates is, that persons suffering from this morbid enlargement receive a slight blow, and this is the immediate cause of the rupture.

**Fibrinous Deposits or Embolic Infarction.**—As we have already said, two different conditions of spleen prevail in heart disease according as the disorder is obstructive or infective. In obstructive heart disease the spleen is dark and hard; it, indeed, partakes in the indurated state which is common to all the great viscera in cardiac ischæmia. In acute endocarditis with ulceration, on the other hand, the spleen is generally soft and large, and it is apt in these cases to show certain local diseases which are known by the term "embolic infarctions," or sometimes "fibrinous deposits." These embolic infarctions appear on the surface as pale patches abruptly circumscribed, and generally situated in the anterior thinner border of the spleen. There may be but one or two, or the number may be very great; their size too is subject to much variety; when cut through they are seen to dip into the tissue like wedges, this shape corresponding to the distribution of the vessels; for if we examine our specimens of the partially injected spleen, we shall find that when the small branches only have been filled, the portions injected correspond in their wedge-like form with the patches we are speaking of. The appearance of the deposits depends on the stage at which they are found; when first formed they are paler than the surrounding texture, and are surrounded by a narrow, deeply-congested zone of tissue, which defines them strictly and plainly. If the artery of the affected part be opened, or its section examined, it will be found to be full of ante-mortem clot. In the course of a few weeks from the time of its first formation the patch grows gradually paler and paler, acquiring at the same time a characteristic yellowish or brownish tinge, and at last the portion shrinks, so that its surface sinks below the general level, and the tissue around it falls in until finally a scar of fibrous tissue may be all that is left. Scars along the anterior edge of the spleen often have such an origin, as we can prove by observing how frequently they are associated with ancient heart disease.

This is the course taken by the simpler examples of embolic infarction, but in some cases more severe results arise. Thus, instead of a tissue going through the above simple wasting, the patch shows signs of inflammatory softening or it suppurates, this change being accompanied by peritonitis of the capsule of the spleen. Such a result we have several times seen in cases of ulcerative endocarditis. When this is the case the same severity of the course of local change coexists in any other examples of embolism that may be present in the kidney, brain, or other organs.

We believe the cause of these infarctions is the entrance of clot into the arteries of the spleen, and that the variation of the action in the part depends on the nature of the clot introduced. If it be clot formed passively in the heart, &c., by simple deposition of fibrin, then the infarction leads ultimately to wasting, but if the embolus is a fragment from an ulcerated spot, then it sets up a septic inflammation, even to suppuration, in the seat it reaches. Micrococci have been found in the vegetations, and described by Dr. Osler and others, which makes the disease a true infective one. We must then distinguish two sorts of result—Firstly, those due to the obstruction of the artery. The consequences of this occurrence are these: the distal part of the obstructed vessel and the vein corresponding become distended, apparently by the vessels suffering malnutrition and losing their power of resisting the general vascular tension; the blood leaks by a retrograde course through the vessels and fills the interstices of the tissue, and thus flooded with extravasated blood fails in its nutrition, and undergoes involution and absorption. The vessels in the borders of the affected part partake of the distension, and cause a dark zone of congestion around the patch. Secondly, the changes due to inflammation are those ordinarily seen in that process, but complicated by the effects of this circumstance, that the inflamed part has its vessels plugged, the result of which is that the inflammatory activity is generally limited to the zone around the plugged part, which is apt to separate in a more or less necrosed state, forming thus an ill-conditioned abscess, into which the living parts around, partaking in the inflammation, continue to discharge pus.

The milder form of this process is what we generally see in ulcerative endocarditis, the severer is identical with what we find in pyæmia; indeed, it may be taken as a description of the ordinary course of pyæmic abscess of parenchyma, which many of the best authorities believe always arises by embolic plugging of the part. When these embolic abscesses form in consequence of inflammation in the left side of the heart they may be well called by the name of *arterial pyæmia* to distinguish the whole case by this title from the more common cases of pyæmia, due to surgical sores and wounds, the difference being that in the latter cases the disease generally affects the course of circulation of the venous blood, while in arterial pyæmia the secondary suppurations are found in the course of distribution of the arterial blood. The mechanism of the local process is, however, the same in both cases. Embolic infarctions of the simple kind uncomplicated with infection are rarely met with except in heart disease. They do, however, sometimes occur, and then it is a question how they are formed—probably by a coagulation of the blood itself, favoured by special chemical changes which have gone on within it.

The microscopic appearance of these infarctions reveals the persistence of the splenic tissue in them, and the gradual disintegration of it by a fatty transformation, while a quantity of hæmatoidin crystals are always formed in it. This presence of blood-crystals, together with the superficial seat and wedge-like shape of the patches, forms the chief means of distinguishing hæmorrhagic infarctions from other local diseases of the spleen such as tubercles, Hodgkin's deposits, &c.

**Inflammation and Abscess.**—In describing embolic infarction we have mentioned the fact that when the embolus comes from an inflamed endocardium into the spleen it sets up inflammation in the embolised spot, the result being that an abscess forms there which has all the characters of position and outline that belong to these infarctions, especially in that they are found at the surface and usually at the thinner edge of the organ, dipping into the tissue in the form of a wedge.

Abscesses of the same general characters arise from common pyæmia, such as we meet with after surgical operations. These pyæmic abscesses have quite the same appearance as the embolic abscesses. Often all the stages of the embolic and suppurative processes can be seen in the same spleen. The earlier stages are exactly alike in the two conditions, so as to suggest it as probable that pyæmic abscesses are also embolic, and take their origin in particles carried from the primary sore; this theory we have elsewhere considered. The abrupt demarcation of a part of the tissue by a deeply congested zone, while the included portion becomes gradually paler and more opaque, is common to both conditions, but soon the pyæmic part softens down into a cavity containing pus, in which are fragments of the original tissue in a state more or less broken down and detached. Some cases show more severe extremes and change than others, in some gangrene may occur; the gangrenous condition is generally dependent on a gangrenous state of the sore or wound that causes the pyæmia.

These abscesses are generally not large, though by their number, and by breaking into each other, they may occupy more than half the spleen. The peritoneum over them is coated with lymph, but it is very rare to see general peritonitis set up in this way.

It is a question whether idiopathic abscess of the spleen ever occurs; we have met with a few cases of long-standing abscess in the organ, but it was doubtful whether they had not an hydatid origin; one of the cases was from a sailor who had recently come from the tropics. Yet the spleen is not unfrequently found involved in suppuration.

Among other cases which are very obscure during life are some which are found to be due to localised suppuration in the left hypochondriac region. Circumscribed abscess in this part we have now seen several times; in some cases it has been due to a chronic ulcer of the stomach. In two or three there has been a communication with the intestine, although whether this was the primary or secondary affection has not been very clear; we have seen also one or two instances of suppuration in this region touching the spleen where no such cause could be found. Lower degrees of inflammation of the spleen are very difficult to recognise; it is not unfrequent to find the peritoneal coat of the organ more or less inflamed, and coated with plastic lymph, and we still more frequently meet with the same process in a later stage, when the spleen is adherent to parts around, especially the diaphragm ; generally no further peritonitis is present, and hence we are almost obliged to conclude that the inflammation belonged to the spleen itself rather than the peritoneum. In this way we may infer splenitis from perisplenitis, but the conclusion is not so satisfactory as if we had obtained direct evidence of inflammation of the tissue of the spleen, for we occasionally meet with perihepatitis, without inflammation of the liver. We have, however, on one or two occasions met with diffused subacute splenitis, the affected portion of the spleen was more solid and deep coloured than the rest of the organ ; and the whole spleen was much swollen and adherent to its bed. This happened in the body of a person affected with syphilis, who had a similarly diffused subacute pneumonia. On the whole the character and means of recognising the lower degrees of splenitis are very unsatisfactory, and stand much in need of further investigation. The condition of the capsule should be carefully observed ; a little recent lymph spread over it easily escapes notice. We have seen such a layer of recently formed lymph on a soft spleen in a case of death from rheumatic fever.

It is probable that under the name of *chronic inflammation* many slow changes taking place in the tissue of the spleen might be included. It must be remembered that the fibrous capsule is elastic and contains non-striated muscular fibre, and that from this, fibro-elastic bands are given off which traverse the organ and support the pulp and glandular contents. Dr. Roy has demonstrated that the spleen undergoes a rhythmical contraction about every minute. Now, we find sometimes the spleen hard and these fibrous bands much thickened. Dr. Goodhart described a case in which the trabeculæ were so enlarged that the organ appeared mainly composed of muscular fibre. Such a case might be called *interstitial splenitis.*

A chronic induration is also observed in heart disease and cirrhosis of the liver, also in the so-called albuminoid spleen of rickets.

Various other changes are met with in the spleen which might fairly be classed under the term *inflammation,* such, for example, as is seen in typhoid, where the organ is large and soft and the corpuscles swollen. In *scarlatina,* Dr. Klein states that all the parenchymatous organs undergo a change. The Malpighian corpuscles of the spleen enlarge, and the appearance of the cells is altered, whilst a hyaline change takes place in the arterioles together with much thickening of the walls in which nuclei are seen.

**Chronic Capsulitis.**—*Chronic thickening of the capsule.*—We have just mentioned the frequent adhesion of the spleen to the diaphragm ; this is generally found quite unexpectedly, but it often occurs in the bodies of persons who have suffered from tropical fevers. We believe such adhesions are an important cause of pains in the left side ; having met many instances in which pain in the splenic region was much complained of by persons who proved to have adherent spleen. The pain is no doubt due to the resistance such a spleen opposes to any cause of distension during portal congestion. Besides these adhesions, the capsule of the spleen rather frequently shows circumscribed thickenings of various sizes, and more or less diffused over the surface, but usually limited to the convex side. These often reach a surprising thickness, and then closely resemble common fibro-cartilage. Section shows them to be laminated parallel to the surface, and the microscope reveals a fibrous structure, the fibres being arranged in dense areolated lamellæ. Such semi-cartilaginous thickenings of the splenic capsule usually occur with similar thickenings of the capsule of the liver, and they may be regarded to be among the evidences of chronic alcoholism that post-mortem examination reveals. A minor degree of the same thickening is occasionally found, marking the surface of the shrivelled spleen in an elderly person.

**Apoplexy.**—We may here mention this form of disease, which proves rapidly fatal in domestic cattle, and has the character of an acute fever, the spleen being found distended with extravasated blood and enlarged to an enormous weight. It is communicable to man as anthrax, and then, as already mentioned, peculiar changes are found in the stomach and intestines. It has a characteristic bacillus.

*Syphilis.*—We have met with a large gummatous mass in the spleen, and in one case this had softened into a fluid.

**Lardaceous or Waxy Spleen.**—We have already spoken of lardaceous liver, and the spleen may be affected in a similar manner; indeed, it is one of the principal seats of the affection. In most cases the amyloid matter is not uniformly diffused, but affects the Malpighian corpuscles, which become greyish and semi-pellucid, so that on section they give to the organ an appearance that has been well described under the name of the "sago spleen," the corpuscles looking like boiled sago-grains scattered through the tissue. On applying tincture of iodine thinly to the surface of such a spleen, these corpuscles become in a few seconds stained of a deep brown colour; this dark colour is the characteristic reaction of the amyloid substance in lardaceous disease.

The size and appearance of the spleen in these cases is so little affected that the microscope is required to reveal the amyloid change, but thin sections soaked in iodine-water show under the microscope the small arteries to be characteristically coloured. In the same sections the state of the Malpighian corpuscles can be more closely examined, and it will be seen that in them the amyloid change is not confined to the small blood-vessels, but has extended from these to the lymphoid structure which composes the parenchyma of the corpuscle, and then to the trabeculæ until the whole structure is converted into the usual glistening mass which readily absorbs iodine, and acquires a characteristic deep brown colour.

Such a microscopic examination by the aid of iodine is always required to discover the lesser degrees of lardaceous disease, for it must be remembered that the customary application of tincture of iodine is but a coarse test. When the Malpighian corpuscles are in the above-described sago-like state, the spleen is not generally large; but sometimes no sago-grains appear, while the lardaceous spleen is large and of a homogeneous appearance on section. As far as we have observed, Malpighian corpuscles are always absent in these uniform lardaceous spleens; when present the corpuscles are always first and chiefly implicated. The consistence of a lardaceous spleen is almost characteristic; its peculiar feel, resembling that of firm lard, will generally be recognised after it has once been carefully noticed. Like all lardaceous organs, such a spleen is anæmic, and has a peculiar translucent and lustrous look on section, is capable of being cut into thin slices, and has a soft toughness, very closely resembling the consistence of raw bacon. It is necessary to remember that lardaceous matter never or rarely occurs free, but that it is most intimately associated with the structural elements, being within the component cells of the arteries, &c., and causing these cells to swell; hence the unfortunate impossibility of obtaining it pure for chemical examination.

**Tubercle.**—Tubercle is met with in the spleen in two forms, first as minute grey granulations, sometimes extremely small, not larger than grains of pepper, at others approaching the size of millet seeds, and then having a more yellowish colour. This form of tubercle is always found as part of a general tuberculosis, and in size, &c., the tubercles correspond to those found in the other parts of the body. When small and grey such tubercles are with difficulty distinguished from the Malpighian corpuscles, but we have observed that a stream of water will decide the question. The Malpighian corpuscles are always softer than the rest of the spleen, and so wash away first. Tubercles, on the other hand, cling firmly to the trabeculæ, while the pulp is washed away.

The other form of tubercle is found in large yellowish deposits scattered about in the organ. It is much more rare than the miliary kind, and its pathology is less definite, cases occurring which present characters more resembling tumour-formation than tubercle, while others belong to syphilitic gummatous products. Precise criteria for the definition of these several conditions are indeed wanting, but in determining the nature of any particular case, we should be guided by these characters as distinctive of tubercle: first, a generally rounded figure of the masses, which vary in size; second, an almost complete caseous transformation of the deposit; and, third, the presence of miliary-sized granulations, forming the recent growing outer edge of the patch, which, if large, may be softening down in the centre.

It is probable that these large tubercles in the spleen are primary, that is, belong to its proper pathology, while the miliary tubercle is always secondary to some other tuberculous source.

**Lymphadenoma—Hodgkin's Disease.**—This name is useful as distinguishing a diseased condition common to the spleen, the lymphatic glands, and often to the liver and other organs, which was first pointed out by Dr. Hodgkin. It constitutes a disease that can be recognised clinically, and is not unfrequent. The glands are found enlarged, and containing the same formation as that in the spleen. The state of the spleen is very remarkable. It is increased more or less in size, and contains a number of deposits of yellowish-white, tolerably firm, opaque matter, resembling at first glance masses of suet, scattered through the organ, or much more rarely collected into a larger mass in it. The masses are generally of angular outline, of very various sizes and embedded deeply in the spleen-tissue. They are connected with the trabeculæ of the spleen, and have once been observed to radiate from its vessels; a few reach the surface, and sometimes a granular formation of the same substance is found in the capsule. The deep seat and the absence of hæmatoidin pigment distinguishes them from embolic infarctions. The structural characters of the patches vary somewhat, but the microscopical appearance generally shows a fibrous meshwork containing more or fewer lymphoid cells, *i. e.* the structure known as "lympho-sarcoma" or "adenoid" tissue, or more roughly as "fibro-nuclear tissue;" sometimes the fibres preponderate and the masses are harder; sometimes the cells are vastly more numerous, and then both in appearance and in tendency the diseased patches closely resemble cancer, so that they may spread to parts around malignantly. But some of the cases graduate towards tubercle, becoming caseous, and softening down, or being associated with general tubercle. Others have much more the character of lympho-sarcomatous tumour.

The association of these formations in the spleen with similar growths in the portal canals of the liver, and the glands, has been already described under "Lymphadenoma."

The relation of Hodgkin's disease to "leukæmia" has yet to be determined. The latter, as already described, is the case where a large number of organs are infiltrated with white cells. They sometimes have a tendency to coalesce, and therefore if they still further formed lymphoid masses, they would approach very nearly to Hodgkin's disease.

**Cancer.**—Excluding cases of the kind we have described under the name of Hodgkin's disease, primary cancer of the spleen is exceedingly rare, and the spleen is also very rarely affected by secondary cancer. When cancer is widely diffused in the body we may find the spleen implicated, masses of growth rising from the surface or embedded in the interior. These secondary masses of cancer have a rounded figure. A spleen in the Museum exhibits within it a melanotic tumour, such as existed elsewhere in the body from which it was taken. The cancer is usually a soft and rapidly growing sarcoma or carcinoma.

**Hydatid and other Cysts.**—Hydatid of the spleen is not common. A case occurred here some years ago, where a very large cyst was attached to the spleen, as seen in the drawing; it was apparently hydatid. Old calcareous hydatid cysts have been met with in the spleen. In a specimen, which has been thought to show hydatid cysts, there are two or three cavities within the tissue of the organ, and thus it appears to be rather a growth of cystic formation resembling that in the liver which has already been shown. Mr. K. Thornton removed by operation a large spleen full of cysts. These were infiltrated with dark fluid and cholesterine as of disintegrated blood.

# DISEASES OF THE SUPRARENAL BODIES

*Addison's disease.*—A destructive inflammatory (or tuberculous?) disease of the suprarenal capsules. The great discovery, which will deservedly perpetuate the name of Addison, has had the effect of directing the attention of the profession to the diseases of the suprarenal capsules. That known by his name, Morbus Addisoni, is characterised by pigmentation of the skin, asthenia, and a very uniform change in the suprarenal bodies. These are usually found somewhat enlarged and hardened; more or less firmly adherent to the parts around them, the surface nodular or uneven, the proper shape being almost lost, and generally no traces of the natural structure observable, their place being occupied by an adventitious material. This material is of two kinds, according to the stage of the disease; the one being a uniform grey translucent substance, and the other opaque, yellow, and friable. The two stages of the disease we have been able to trace in different cases. In the one having a short history, and in which we have every reason to believe that the disease is recent, the organ has been found two or three times its natural size, and consists of a firm solid substance of a greyish-white colour, somewhat resembling lardaceous material. It is of an albuminoid nature, surrounded by a thick capsule which has quite destroyed the organ. In the second stage, seen in the more chronic cases, this albuminous material has in parts undergone degeneration marked by a number of yellow spots scattered throughout it. These increase in size until large, round, yellowish masses are seen embedded in the original translucent substance, and later on, the whole of the latter has disappeared, leaving nothing but the yellow material. It is in this stage that the disease is usually found, and is then often styled tuberculous. The microscope shows in the more recent parts lymphoid cells and fibres, and in the latter yellow masses, granular detritus, and withered cells. At a later period, which might be styled the third stage, the degeneration has proceeded still further, so that a general shrinking has taken place, leaving nothing but a little cretaceous matter; or instead of this softening having taken place only a little pus-like material is found enclosed in a capsule. This is the explanation of those cases where it is said that the capsules had entirely disappeared, and nothing but a little purulent matter was found in their place; or, what is more common, a little cretaceous matter together with a few drops of pus. In time even this softened matter may disappear, and then it is absolutely true that no trace whatever of the capsule can be perceived. Several cases have been recorded of well-marked Addison's disease where not a vestige of the organ, healthy or morbid, could be discovered. We are aware that some observers object to this explanation of their absence, but we think the cause given is quite adequate, especially when all the symptoms of Morbus Addisoni had been present. There is nothing in the microscopic appearances of the disease usually found to denote anything more than a degenerated albuminous product. The term tuberculous has been given to it, but there has been nothing to warrant this from the presence of tubercles elsewhere, nor do we think characteristic bacilli have been found. We have regarded it merely as a caseous degeneration of an inflammatory product, and this is somewhat corroborated by the fact of the disease having been found in several cases secondary to adjacent caries of the spine. With our present knowledge the term inflammation best covers its pathology, for it makes Addison's disease correspond with the primary affections of other organs, as some forms of phthisis, cirrhosis of the liver, Bright's disease, or sclerosis of the nerve centres. It is quite comparable with these in its being primary and existing alone. It is comparable also in the fact that in studying the diseases and attendant symptoms of most organs we do not take into consideration those which are due to the accidental invasion by some morbid process from without like cancer, but only those which are intrinsic or have commenced from within. Nothing, indeed, could be predicted of what the symptoms might be in the case of a cancer of the liver or kidney, and in the same way nothing distinctive could be stated regarding a cancerous suprarenal capsule. Already a large number of cases have been observed where the adrenals were attacked by various adventitious diseases, but in none were the symptoms of Addison's disease present. This is what might have been anticipated. It might also be noted that the absolute destruction of the organs corroborates the position we have taken of the disease being primary, for nothing is known of an adventitious growth such as cancer, first destroying an organ, and then itself altogether disappearing, whereas in the chronic inflammatory process such a complete atrophy might occur; the liver and kidneys are seen to waste until that stage is reached which is just compatible with life, and there is no reason, could their functions be adopted by another organ, why they should not waste still more, but in a non-vital structure like the suprarenal capsule the process might still go on until nothing of the organ is left. This we think really occurs.

When the organs are much enlarged, and especially when an inflammatory process takes place around them, the semilunar ganglia, and the nerves proceeding from them, are apt to be involved; and we are then able to trace these nerves quite into the nodules of the diseased mass. This has given rise to the question whether the symptoms of Addison's disease are not due to the implication of these important sympathetic nerves rather than to the destruction of the organs themselves. It is clear that these are not vital in the ordinary sense of the term, since it is certain persons can live for years without any portion of their structure remaining. Addison was aware of this fact, although his statements were attempted to be refuted by showing that animals could survive after the removal of their adrenals, and on the first occasion of his bringing his discovery before the profession he suggested the nerve theory in these words, "We know that these organs are situated in the immediate vicinity, and in contact with the solar plexus and the semilunar ganglia, and receive from them a large supply of nerves, and who could tell what influence the contact of these diseased organs might have on these great nerve centres, and what share that secondary effect might have on the general health and in the production of the symptoms presented ?"

*Apoplexy.*—The organs may be found sometimes distended with blood; the tissue being very soft, probably any slight cause giving rise to extravasation of blood would soon flood the organ.

*Tubercle.*—The adrenals may be found to contain tubercle, in common with other organs in general tuberculosis.

*Lardaceous disease.*—This is found very often in connection with lardaceous disease of other organs, as in tertiary syphilis.

*Fatty change.*—It may be remarked that the term "capsules" given to these organs was owing to the fact that they were often found hollow in the interior. This was from the breaking down of the softened tissue. A section of a healthy organ shows a white material in the middle with a vein running through it, surrounded by a brownish or yellowish cortical substance, the latter is composed of rows of large cells. Now, all these parts may become exceedingly fatty and soft, so that the organ is easily broken up and crushed in the attempt at removal.

*Adenoma.*—We sometimes meet with small growths or tumours on the surface of the adrenals. These little projections are composed of true glandular structure, and remind one of the analogous supernumerary spleens.

*Cysts* are occasionally met with in the adrenals, hydatids rarely.

*Cancer.*—Cancer of various kinds may be found growing from or invading the adrenals. We have seen one case where a large part of the organ was involved, and the growth appeared to be primary. In the same way we may occasionally meet with *sarcomatous* and *melanotic* growths. We think too in a case of syphilis we once examined that a deposit in the organ was of a gummatous nature, and a similar case has been published by Dr. C. Turner.

It has been stated by Stilling that the adrenals undergo a *compensatory hypertrophy ;* that is, if one of them be removed in a living animal the other will grow after the manner of the kidneys.

# DISEASES OF THE URINARY ORGANS

## KIDNEY

**Malformation.**—The commonest abnormal condition is the *absence of one kidney;* sometimes there is no trace of it, or its ureter. At other times some of the remains of the tissue are left; the latter fact suggests the same cause for its occurrence as for numerous other malformations already mentioned—that is, a morbid condition during fœtal life affecting the nutrition of the organ and causing its subsequent atrophy; for in these cases we find remains of the blood-vessels, and the ureter may be present, though extremely small. The suprarenal body does not share in the malformations or malpositions of the kidney. The kidney on the opposite side is enlarged; so that it is often equal to the natural size of both kidneys. Sometimes the two kidneys are united together over the spine, constituting the *horseshoe kidney.* The junction is always at the lower part, so that the semicircular organ has its convexity downwards, the blood-vessels enter on each side as usual, and the ureters pass down in front of it. The union of structure may be complete, but in one case we met with a junction by merely fibrous tissue. The kidneys may also become altered in shape by a curvature of the spine.

A very common abnormal condition of the kidneys is that in which they are *lobulated*, a state which is natural in the fœtus; the surface is seen to be deeply fissured and divided into distinct portions.

In one example in our Museum both kidneys are united to form an unshapely mass, which lay within the pelvis. It has two ureters; but in some cases the two kidneys are united over the promontory of the sacrum, and there is a single ureter extending down and opening into the bladder in the median line. The vessels in these cases of low position of the kidney come off from the lower part of the aorta, or from the common iliac. Kidneys placed thus low may appear as tumours during life, and give rise to conjecture and apprehension.

Cases have been met with where a cord has been found running near the ureter. This has appeared to be the remains of Müller's duct. Below, it has terminated in the bladder, and above has passed into the position of the Wolffian body.

**Malposition.**—This may be congenital or acquired. It sometimes happens that the head of the right kidney is dislocated from its natural position, wherein it is insinuated between the liver and the diaphragm, making a shallow pit on the hinder margin of the liver. This displacement happens generally in women, as if through tight lacing, but it is often directly attributed to a strain. We have seen several examples of movable kidney, and in all these the kidney had turned so that its long axis was transverse and antero-posterior, the upper end projecting forwards between the colon and the liver. By pressure such a kidney may be made to cross in front of the spine, yet it is altogether behind the loose peritoneum, and seldom has anything like a mesentery. A meso-nephron has, however, been described. The displaced organ is said to have compressed the colon or vena cava, but there was no ill result in our cases. A movable state of the left kidney is much more rarely seen.

The position of the organs may be irregular in other ways; thus, occasionally, one kidney has been found situated low down on the psoas, or over the promontory of the sacrum. We have seen two kidneys on the right side and none on the left.

**Hypertrophy.**—It is generally true that when enlargement of one portion of a viscus does take place, it is merely compensatory for the atrophy of another. Thus if we find one kidney much larger than natural, we discover the other correspondingly small. These organs, however, may be often larger and heavier than usual, with evidence of congestion, in fever, pneumonia, &c. Or the enlargement may result from over use; at least, we have several times found healthy kidneys to weigh as much as eighteen ounces in persons who have drunk enormously of beer; also in diabetes the kidneys are generally fourteen or fifteen ounces, that is, they exceed their proper weight by one half.

**Atrophy.**—The most common cause of general atrophy of the kidneys is chronic Bright's disease, an example of which we had not long ago, where the kidneys together weighed less than an ounce and a half. Sometimes we find one kidney wasted, or scarcely a trace left, as we just now mentioned. We have met with some instances of small atrophic kidneys in young persons where the organs appeared to have been congenitally imperfect. Thus, in a young man of twenty-three years who died of chronic albuminuria there were but two pyramids in each kidney; and the kidneys themselves were very small, but without that granular and contracted appearance which usually characterises the small kidney in ordinary chronic Bright's disease; they were evidently deficient in development. We have seen degrees of this atrophic state so many times that we believe such a congenital deficiency to be an important predisposing cause of chronic insidious disease of the kidney in early life. We very often find local wasting of the kidney, its outline broken by gaps, in the depth of which fibrous tissue is found; some of these are evidently from old embolism, others from old inflammation and suppuration around the kidney, for not only may traces of such be found, but the subjects of the affection are commonly those who suffer from stricture, and are likely to have had nephritis; indeed, we have often had thus very sufficient proof of recovery from nephritis caused by stricture or stone in the bladder. Wasting may also occur from impacted calculus in the kidney.

**Injury.**—Slight lacerations of the kidney may heal, especially in those cases where the peritoneum has not been injured, and any effused blood has been confined to the cellular tissue of the loin. Fractures of the kidney generally run in a direction vertical to the surface; in the course, that is, of the radiating pyramidal structure of the kidney. Hence there is often little bleeding, since the large vessels, having the same direction, are separated and not torn across. The urine, too, which is a most irritating fluid, scarcely escapes, and not in sufficient quantity to set up inflammation around; at least we are constantly examining the bodies of persons who have died several days after severe lesions of the kidney with extravasation of blood, without finding any products of inflammation. Sometimes the kidney is broken across, and its pelvis or ureter torn; in such a case there may be a simultaneous rupture of a vein, and thus much blood may pass down the ureter; perhaps coagulating within it, and forming moulds of its cavity, which escape in the urine, by which moulds this accident was on at least one occasion recognised by Mr. Hilton during the patient's life.

**Inflammation of the Kidneys.**—All are familiar with the wide difference between inflammations that are severe enough to destroy the structure of an organ, and those milder degrees that constitute catarrhs and inflammatory induration. In no organ is the distinction between these two branches of inflammation more important than in the kidney, or more evident. The destructive inflammations were known long ago, but it was left for Bright to discover and interpret the less obvious inflammations.

*Suppurative nephritis.*—This is the nephritis formerly so called when Bright's disease was unknown. It produces tangible results in the way of suppuration; and during life its effects on the urine, except so far as they are hidden by quantities of pus from the bladder, are too evident to escape notice. The urine usually has in it unmistakable inflammatory products. But it is a secondary affection, that is, it never results from any of the forms of Bright's disease or other diseases which belong primarily to the kidney structure. It is always traceable either to inflammation of the bladder or to pyæmia, and hence it is often called the "*surgical kidney,*" to distinguish it from Bright's disease, which comes under the care of the physician. Such a term is, however, not absolutely distinctive, for in the medical wards inflammation of the bladder often terminates in suppuration of the kidneys; especially the cystitis resulting from paralysis in spinal affections. In such cases as these, or in paralysis from fracture of the spine, or else in the widely different conditions of urethral stricture, calculus, or enlarged prostate, &c., the bladder becomes inflamed. This happens through retention of the urine until it acquires irritating properties, while the necessary catheterism adds to the irritation; the inflammation runs from the bladder up the ureter to the pelvis of the kidney; then an extension of the purulent process occurs until a suppuration arises in the cortical part of the organ. The mechanism of the extension is sometimes obscure; thus, we may find the ureters not evidently affected, or but slightly so, and they generally are not nearly as much diseased as the tissue of the kidneys. Hence some suppose that bacteria or some other means of conveyance carry up infection to the kidney.

If a patient has died with any of the diseases we have named we very usually find the kidneys much enlarged, and their surfaces more or less covered with minute points of pus, or small abscesses, and a section may show the suppuration to run down through the cortex of the organ, reaching to the cones, and often we can trace it on through these; but in cutting sections with this object we must bear in mind the waving course of the tubes, and then we shall perceive that what appears on a section to be a deep-seated isolated suppurating patch is only the continuation of a superficial patch, down through towards the pelvis of the organ; a fortunate section will perhaps show the suppurating tract in nearly all its length through the cones and cortex, and thus the distribution of the disease plainly traces the history of its spread from the pelvis of the kidney upwards. The abscesses rarely reach any size, so as to form a collection of pus beneath the capsule; and it is very rare for the peritoneum covering the kidney to be involved so as to set up a peritonitis. In all these cases the mucous membrane of the renal pelvis is inflamed in the same way as the urinary passages below, constituting *pyelo-nephritis.*

Suppuration of the kidney is a very grave addition to the danger of cystitis, but there is no special symptom by which it can with certainty be recognised in the presence of the already existing disorder of the urine; it has to be inferred from the gravity of the general condition.

The renal pelvis may, however, be inflamed without the substance of the organ being necessarily implicated. The mucous membrane is red, vascular, covered with a muco-purulent secretion, and not uncommonly by flakes of adherent lymph. For this inflammation the term *pyelitis* is used.

It happens sometimes that the surface is more than usually involved, and the capsules of the kidneys, with the adjacent parts, may be those especially inflamed; to such a condition the term *perinephritis* has been given. In acute cases of this kind we find the capsule highly vascular, while pus forms around it, producing abscesses which may seek an opening in the loins or into the colon, &c. But it is more especially as a chronic affection, or as a result of a former inflammation, that we would direct attention, because it is often overlooked. We find, for example, on attempting to take out a kidney, that it is firmly fixed in the loin, so that the organ often slips out of its capsule, which is left *in situ*, very much thickened, and having much indurated cellular tissue on its exterior, closely involving the adjacent fat; the surface of the kidney itself is rough or puckered, and perhaps torn by its separation.

Perinephritis, both acute and chronic, is sometimes caused by blows or other injuries to the loin, but it is mostly found in the same class of cases where the acute suppuration exists, viz. cases of stricture, &c.; suppuration, however, is occasionally found around the kidney as apparently a *primary* affection. There can be little doubt that the adhesion indicates a former inflammation of the organ, and from the contraction of the substance of the kidney it is possible that even pus has sometimes been formed. Such a process probably accounts for the wasted kidneys sometimes met with; thus, in cases of stricture which we have examined the kidneys have been much wasted, both organs presenting localised wasting, distributed so like the nephritic suppuration in stricture cases, that no doubt it arose in attacks of nephritis which had occurred several years before.

In cases of *general pyæmia*, especially those caused by periosteal abscess, small deposits of pus may be found in the kidney, as well as in other organs. We have noticed also that in cases of pyæmia from perineal section or lithotomy, &c., the kidneys show a greater proportion than usual of abscesses, although no sign of pyelo-nephritis be present. This fact is interesting, showing, as it does, that organs already having a partial cause of inflammation fall more readily into pyæmic suppuration. Such facts are not sufficiently borne in view by those who regard all pyæmic abscesses as embolic in their origin.

Abscess of the kidney is generally tuberculous, and we shall speak presently of this form of disease.

**Bright's Disease.**—It was known before Bright's observations that the urine is albuminous in some forms of dropsy, but he first demonstrated that this albuminuria is due to disease of the kidneys. Such kidney diseases as produce albuminuria have ever since been called by the general name of Bright's disease; and although the renal changes which agree in producing albuminuria present, as we shall see, several distinct varieties, yet the characters which they possess in common unite them into a practically well-recognised kind. It is not so very surprising, as at first might appear, that these states of kidney should have long escaped notice; for the kidney itself gives usually no pain, and the urine is clear, while death occurs under widely various circumstances of dropsy, acute inflammation, apoplexy, &c., not one of which points evidently to the kidney as its source. The discovery and proof of the common cause of the vast number of different disorders produced by this disease will ever make Bright's name illustrious.

We have said that the changes in the kidney which produce Bright's disease are various, but they are not so many as the crowd of names proposed for them ; it is unfortunate that so very many terms are employed in speaking of this disease, terms which are incongruous and cannot be brought to correspond with each other. Some names regard the general appearance of the kidney ; thus, we constantly hear of the chocolate kidney, the mottled kidney, the large white granular, or contracted kidney, the lardaceous kidney, &c. These terms are simple and explain themselves, but others are more ambitious and serve to embody theories of the nature of the disease often taken from disputed analogy or doubtful microscopical observations, so that they conflict more or less, and perplex the inquirer seriously. Thus, the names "*acute desquamative nephritis*," "*tubal nephritis*," "*acute diffuse nephritis*," "*croupous nephritis*," are all used for the same disease by distinguished authors, respectively by Drs. George Johnson, Dickenson, Delafield, and Niemeyer.

The plan which the College of Physicians in its nomenclature has adopted, of calling all those conditions, so open to dispute, by the general name of acute and chronic Bright's disease, is that which we have always followed ; it is clinically convenient, and involves no doubtful theories.

Bright himself had already recognised this great distinction, and was aware that he had before him two well-marked types of disease. In the case where much dropsy was present he found not only albumen in the urine, but a distinct and definite history of illness, and after death an *enlarged white kidney*, whilst in the case where there was albumen and no dropsy there was no history indicative of the duration of the disease ; it was essentially chronic and insidious in its character, and after death he found a *small granular kidney* associated with lesions of the heart and arteries. Since Bright's time, writers, especially German, have described various stages of diseased kidney passing from the acute inflammatory to the chronic form. Thus the great distinction which Bright had made appeared often to have been overlooked, and it was to enforce this distinction that we, many years ago, gave a description of the disease as it was taught by the physicians at Guy's Hospital.* In the main facts the distinction still holds good, although it must be admitted that the portraiture was painted in too strong colours, since it does seem true that we occasionally met with a granular and atrophic kidney which has originated from an acute nephritis. The difference of opinion in these matters still obtains; German writers as a rule, favouring the unity of the disease, whilst the English usually distinguish the acuter forms of nephritis from the chronic, the latter more especially receiving the name of Bright's disease. Those who hold the former view refer to a third kind of kidney described by Bright, which he called the mottled kidney; an organ somewhat large and white, and at the same time granular. This they place in an intermediate position.

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* Mais c'est Samuel Wilks qui en 1853 ('Guy's Hospital Reports'), se prononca le plus énergiquement contre la théorie de l'unicité qui considerait les diverses formes du mal de Bright comme des stades différents d'un process pathologique identique. Pour Wilks il existe deux formes particulières d'alterations rénales: le "*large white kidney*" (gros rein blanc) et le "*small granular kidney*" (petit rein contracté) qui constituent le terme extreme de deux maladies essentiellement distinctes.

Wilks prétend que le clinique n'a nullement démontré le passage de la deuxième forme de Frerichs à la troisième ; au contraire, dil-il, ces deux formes possèdent des caractères clinique absolument distincts.

Le gros rein blanc présente un début brusque ou insidieux, un décours en quelque sorte aigu, marqué par des manifestations assez graves, parmi lesquelles domine surtout l'hydropsie.

Of late the question has much turned upon the morbid histology of the kidney, so that at one time it was maintained that the large white kidney, resulting from acute disease, was an affection of the tubular structure, whereas the small granular kidney was essentially an affection of the interstitial tissue; the one form was therefore called *parenchymatous nephritis* and the other *interstitial nephritis.* At the present day there are those who, admitting these two forms of disease, maintain that they both have their origin in the epithelium of the tubules, denying the very existence of any fibrous matrix apart from the tubes and vessels; whilst there are others who not only assert that the granular kidney is an interstitial disease, but that acute nephritis, or some forms of it, are also due to an inflammation of the interstitial tissue; this being more especially the case in scarlatina and some specific fevers. They maintain that the principal changes in this disease are in connection with the blood-vessels and tissue between the tubes, and more especially in the capsule of the Malpighian body. Klebs and his followers have designated these cases *glomerular nephritis.*

There seems then to be not only different opinions with regard to the relationship of these two forms of kidney disease but to the primary morbid process which starts them into activity. It is admitted, however, by the best recent observers that in acute nephritis changes may be found going on both in the tubules and in the capsule and interstitial tissue generally, the particular structure involved varying in different cases. Also they are agreed that in the small granular kidney the connective tissue is increased, making the organ hard and withering up the tubules and Malpighian bodies. The mere discovery, therefore, of what particular tissue is affected is not sufficient to determine the question as to the distinction of the two forms of disease.

*Acute Bright's disease.*—Typical acute Bright's disease occurs as an independent ailment, associated with albuminuria and dropsy, often caused by cold, and then usually during exposure while the person is

Le petit rein contracté est une maladie essentiellement chronique dont les symptomes sont nuls ou obscure; souvent elle ne se révele même pas pendant la vie.

Le premier se recontre plus specialement chez les jeunes gens, le second est plus propre aux vieillards, où il nait sous l'influence de la goutte, du rhumatisme ou peut être par le fait pur et simple de l'evolution de l'âge ('Pathologie des Nephrite chroniques,' par le Dr. Paul Snyers).

hungry or fatigued. It is often called *acute renal dropsy* or *acute albuminuria.*

But besides the independent disease that would thus be universally called acute Bright's disease we meet with a similar condition as a result of *scarlatina.* About the end of the second week of scarlatina the urine is apt to become albuminous; this may pass off without inducing serious effects, but sometimes, especially if exposure is allowed during convalescence, acute renal dropsy sets in, and when once established is apt to run the same course as, or perhaps a rather more favorable course than, acute renal dropsy from exposure to cold. Renal dropsy thus arising after scarlatina, easily allies itself etiologically with acute renal dropsy from exposure, if we believe that a chill to the desquamating skin is the cause of scarlatinous dropsy. There is an opinion, however, that there is a *primary scarlatinal nephritis;* an inflammation of the kidney being the only way in which scarlatina shows itself, and the same is held of diphtheria.

But it is not only in scarlet fever that albuminuria occurs, it is very apt to appear in other severe febrile conditions, such as typhus, enteric fever, pneumonia, diphtheria, and many others. But it is remarkable that although albuminuria is frequent in these diseases, yet renal dropsy very rarely results from them. Also in chronic exhaustive diseases near their fatal issue, albumen may appear in the urine.

The mere *congestion* of the kidney, causing albuminuria, which accompanies many fevers, or is found in fatal chronic exhaustion, and which does not lead to dropsy, is altogether eliminated from acute Bright's disease by some German and American writers, these several states being grouped under the title "*chronic parenchymatous degeneration.*" We think it is well to recognise strongly the distinction between this class of cases on the one hand, and those that prove obstinate and lead to dropsy on the other. But the name "*chronic*" and "*parenchymatous*" degeneration appears rather gratuitous for application to acute and temporary conditions probably caused by blood change. We have not ourselves found it a frequent thing for the kidneys in old cancer cases or other long-standing cases of exhaustion to present any more degeneration than belongs to the muscles and other parts which waste in the general wasting, so that we do not think this term "chronic parenchymatous degeneration" represents any improvement on the old way of recognising the albuminuria of fevers, &c., as due to "*congestion*" or stagnation of blood in the kidneys. Such febrile *congestion,* however, has other factors besides the mere mechanical obstruction which is found in heart disease, &c., and then is also called congestion. For the blood in fevers is itself greatly altered, so that it stagnates in the skin as eruptions, or it even bursts out, causing ecchymoses; and in these hæmal changes the kidney, doubtless, has its share of disorder. But Dr. Murchison clearly showed that albuminuria in typhus, fatal by convulsions, is due to a severe and evident congestion of the kidneys.

Under these circumstances of *febrile congestion* of the kidney the organ is found large, soft, and of a dark colour ; on section the blood drips from it in greater quantity than natural. The microscope shows the epithelium cloudy, granular, or disintegrating in parts, and in places disturbed from its position, while in the channels of the tubes lymphoid cells are exuded, entangled, perhaps, in fibrinous casts ; these are especially seen in the straight tubes. The interstitial tissues are quite natural, and free from inflammation, or swelling, or fat, or fibrous changes. Thus the whole appearances are not those of a destructive disease of the kidney, but rather of a temporary disorder, corresponding to the quick recovery which follows if the fever is cured. Meantime the obstructed state of the tubes is a source of danger, and if it be extreme may lead to fatal uræmia and convulsions. The state of the tubes is not equal ; some we shall find much more free and healthy than others.

It is of great importance to know what conditions of kidney are implied by such casts in the urine as are recognisable by the microscope during life, and whether such conditions are recoverable. In febrile congestion we find casts composed of lymph-corpuscles, blood, and renal cells, held together by an albuminous product, and such casts when found in more chronic cases signify an acute stage in the particular tubes they have come from ; although in these chronic cases there will be also other casts from neighbouring tubes in different states, casts containing oily grains, fine granular casts, &c. In either acute or chronic conditions clear " hyaline " casts of fibrine may be found ; these are often assumed to come from tubes that are denuded of their epithelium, but we have always found them lying in tubes with the epithelium perfect all around them.

Such *febrile congestion* is a long way from the *acute idiopathic renal dropsy*, yet there is a strong link between them in *scarlatinal albuminuria associated with dropsy.* Some regard scarlatinal disease of the kidney as a part of the general exanthematous disturbance proper to the fever, the kidney being affected like the skin and throat. Others look upon it as congestion of the kidneys caused by exposure of the recently peeled and tender skin to chills. The first view would approximate scarlatinous renal disease to other febrile disease of the kidney, while the second would make it identical with the acute renal disease from cold.

Hitherto the scarlet fever kidney has been taken as the typical and most common example of acute nephritis, for death in acute renal dropsy from other causes is comparatively infrequent. Of late, however, as we have already said, it is maintained that there is an essential difference between the nephritis resulting from scarlatina, and that from cold, the one being interstitial and the other generally tubular. It is true that in outward appearance no great difference is discernible.

The first description of the scarlatina kidney by Klebs, confirmed by Klein, Greenfield, and others, states that the Malpighian body is the part first affected. The capsule is made up of a fibrous envelope lined with epithelium, and in its interior a tuft of blood-vessels. The whole glomerulus is swollen and covered with nuclei; these are also seen in the walls of the capsule itself. Cohnheim also found the capsule filled up with this nuclear growth; at the same time white cells are seen in the tissue between the tubules. This, then, would constitute an interstitial nephritis. If this be so there can be no doubt that there is a slight affection of the tubules at the same time, as in the catarrhal tubular nephritis. Greenfield goes so far as to maintain that in all forms of Bright's disesse, both acute and chronic, the most important seat of the changes is in the glomerulus, although most marked in scarlatina; for in the large white kidney a fatty degeneration of the Malpighian body is present, and in the small granular kidney the same bodies are atrophied. In scarlatinal nephritis of a few days' standing we may see the tuft in the capsule covered by a mass of cells originating in the endothelium which is over it, and Klein, in a most elaborate description of the changes found in the body in scarlatina, states that in the kidney the glomeruli are covered with nuclei, and that hyaline changes take place in the walls of the minute arteries. He also found cloudy swelling and granular changes in the epithelium of the tubules.

There seems, then, a very general acceptance of Klebs' statement, that scarlatinal nephritis is essentially a glomerular inflammation, although all do not go so far with him as to assert that there is no interstitial change or proliferation of epithelium. When the glomeruli are markedly affected they may be seen by the naked eye as round white dots.* If these changes are as we have described, we must recognise two forms of *interstitial hyperplasia*, according as the inflammation occurs around the glomeruli or between the tubules.

In contrast to this form is the *tubular* or *catarrhal nephritis*, where the primary changes are seen in the epithelium, the cells being swollen and granular, or undergoing the change styled by Virchow cloudy swelling; then a crowding of the tubules with lymphoid cells, blood, and casts; these going on to fatty degeneration until the tubes are completely blocked, and a general degeneration of the tissue ensues.

There can be no doubt that in all forms of Bright's disease, both acute and chronic, all the tissues of the kidney may be affected, however true it is that the starting-point is in one tissue alone.

In the acute form, of about three or four weeks' standing, we find the kidneys swollen and rounded, rather softer than natural, and oozing blood freely on section. So far, it is like the febrile congestion we have described; but if we look closely we shall detect a difference in that the texture is more opaque, especially in places. This opacity is hidden a good deal by the amount of blood present; hence it is better seen if the organ be soaked a few hours in water. However, there is less blood in the organs than in febrile congestion; we see on the section that the cortex is most affected; its Malpighian bodies appear as bright red dots. We think the great amount of blood present is chiefly due to the mode of death, by convulsions or coma. In one case of death by febrile exhaustion and asthenia in pyæmia with acute renal dropsy, the kidney showed little congestion, the change then was patchy opacity of the epithelium of the renal tubes, very well seen after soaking in water. This opaque white state of the tubes is, indeed, the essential part of many forms of the disease, and in doubtful cases it is a good practice to soak the organ before deciding as to its presence. The capsule is thick and opaque (but before setting this down to inflammation we must look at other fibrous membranes, and we shall find that they all are thick and opaque through the dropsy); it strips off easily, and the surface of the kidney is then found smooth. The size of the organs and the mottling of them with white subopaque deposit are the essential morbid changes. The whiteness of the deposit is not like the whiteness of anæmia. It is due to fine grains in the renal epithelium reflecting white light; so it is of a creamy appearance, while anæmic whiteness gives the more pellucid appearance of the almost transparent renal epithelium. It is not evenly distributed, but some tubes are much more affected than their neighbours. But in this early stage, although the disease is essentially due to a whitening change, yet the amount of blood present, prevails so as to cause the colour of the organs to be at first sight of a deeper red than natural.

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* 'Path. Trans.,' vol. vi.

When, however, death occurs later in the disorder, as after three or four months, the kidneys are pale, even much paler than natural, while they still keep their large size. To understand why this is, we must remember, first, that the patient's blood has been much reduced by the disease; and, secondly, that the renal tubes have become so swollen by accumulation within them that they compress the capillaries and keep them nearly empty, so that the blood-redness is thus reduced. But meanwhile the fine grains of fat have increased in amount, and, by reflecting the light, like any other white powder, have given more of the creamy opacity before spoken of. But the amount of fat-grains differs in different parts, and so some spots are very white, while around these the colour is greyish or pinkish and less opaque. The more opaque spots are usually small; they occupy the course of bundles of tubes, for they are the fat epithelium of the worst affected bundles, while neighbouring bundles are relatively free. Hence this stage or degree of acute Bright's disease shows itself as a pale kidney. The white opaque spots were the "granules" of Bright, Rayer, and the earlier observers, and this form was their "granular kidney," but the name granular is now given to the tangibly granular, minutely-nodulated kidney, formerly called "contracted" by Bright. These earlier authors thought the white granules a new matter effused in the parenchyma; but Bowman's discoveries were not then made, and the structure of the kidney was not known; the whiteness is not a new matter between, but a change of colour in, the texture, due, as we have said, to the powder-like white particles of fat crowding the renal epithelium.

Yet later, the progress of the same changes continues to make the organs whiter and even larger, until the classical "*large white kidney*" is fully developed. The surface is still smooth. This extreme of whiteness is due to the greater quantity of fat, and the greater and more general swelling of the tubes which expels the blood more than before. Thus we see the whiteness is not due to passive anæmia, since there is a tendency to congestion, which it is important to notice and which reveals itself in three ways—first, the pyramids are deeply congested; secondly, there are often effusions of blood into the renal tubes, forming red spots on and through the cortex; and thirdly, the veins of the surface are engorged, their stellate and arborescent figures being often very beautifully conspicuous. The loading of these superficial veins proves the fact of pressure within the cortex of the kidney, for it is the compression of these penetrating veins which causes its whiteness.

These *congested*, *mottled*, *conglomerate*, and *large white* conditions are thus successive stages of acute Bright's disease. They are not at all distinct kinds of the disease, and by the accidental excess or deficiency of blood much variation of the appearance in any of the stages is induced.

The microscope shows in the large white kidney a considerable degree of change, but yet it strikes one as remarkable that there is not more to account for the tenacious obstinacy of the disease. There is no destruction, as in phthisis; already with the naked eye we can see that the general structure remains unaltered. The disease, indeed, is founded in very minute disarrangements, so that the microscope may disappoint our expectations if we look for anything obvious. The chief changes are in the contents of the tubes. The epithelium is cloudy and opaque in the more translucent parts, and quite opaque in the more opaque white parts, of the kidney; we see tubes in these different conditions side by side. The regularity of the epithelium is disarranged, and some cells are detached, others disintegrated. The central channel is narrowed, or in some tubes obstructed, by fibrin mixed with blood-cells, lymphoid cells, defaced epithelium, &c., but such obstruction does not affect all or most of the tubes. The tubes have been described as denuded of epithelium by desquamation, but we think this must not be too hastily allowed, for in making a section, however carefully, numbers of the tube-sections are emptied, and a single waving of a section through water will empty numbers more. If thickish slices be observed the deeper tubes appear to us to have their epithelium in them, and in very thin sections it is impossible to say that emptiness of the tubes is not through the process of preparation. The names "acute desquamative nephritis," "tubal nephritis," "croupous nephritis," &c., given to acute Bright's disease, rest on the belief that the epithelium is the special structure affected. Nevertheless, we have always found in the conglomerate and large white kidneys that the intertubular tissue is coarse and fibrillar, and often loaded with fat-grains; to make the observation we must cut thin sections and wash out all the contents from the tubes by agitation in water. We have already said that in other cases, more especially scarlatinal, the Malpighian bodies are the parts chiefly affected; they appear as minute white points, and they are found stuffed with cellular formation. The general analogies of acute Bright's disease in comparison with other diseases would perhaps be best expressed by the name *acute catarrhal nephritis;* its intense obstinacy is, no doubt, due to the impossibility of clearing the long tortuous tubes from catarrhal products. "Casts" are only found in some, not all the tubes; they are best seen in the straight tubes of the pyramids; they fill some of these tubes, which otherwise show comparatively little disease. These casts appear to us to arise by the semi-solid matter that obstructs the tubes filling them up, and projecting by a sort of overflow from the tubes, until having reached a certain length they break off.

*Chronic Bright's disease.*—Under this general name several distinct disorders may be brought. Firstly, the later stages of the acute affection which we have just described, namely, the large white kidney, and also the fatty and lardaceous kidney. But the typical and proper chronic Bright's disease is the *granular kidney.* This is in the large majority of cases chronic and insidious from the first, being at no period acute, like the large white, nor is it a mere part of a general lardaceous disease in the abdomen, like the lardaceous kidney. We have already mentioned that Bright called our conglomerate kidney by the name of granular, and gave the name contracted to that kind of kidney we are now concerned with. In its typical form it occurs in circumstances totally different from those of acute Bright's disease. The patient dies from apoplexy, or acute pericarditis, or succumbs, as he otherwise should not, to some intercurrent accidental affection, there being no dropsy present, nor, indeed, anything to direct attention to the disease of the kidney if the urine should escape examination.

In this form of Bright's disease the tendency of the kidneys is to waste, so that they may be reduced in size until they weigh, as some do, only one ounce and a half. We generally find the kidney much smaller than natural, and with its capsule thickened and adherent. The surface not smooth, but on removal of the capsule seen to be irregular and minutely nodulated with small projections, which are now referred to in the title "granular" applied to the kidney. The general figure of the kidney is retained, but numerous irregularities of surface much deface its form. Its colour in a typical uncomplicated case, that has given no symptoms during life, is often not different from that of a healthy kidney, but it may be finely mingled with whitish, or greyish, or yellowish-white, through lodgment of fat in the tubules;

when this is so the granular outline and strange colouring make the kidney look very morbid. Complication with acute Bright's disease we shall see has the same effect. Section shows the cortex to be thinner than natural; it may be extremely thin; it is not only thin, but its structure is seen to be confused or lost, so that the natural radiating striated appearance is not perceivable or is evidently destroyed; minute cysts may be seen in it more or less numerous. The masses of cortical tissue between the pyramids are less wasted; the pyramids themselves are not wasted at all. They are large—some think hypertrophied vicariously—and may show small chalk-like dots, which are minute calculi made of crystals of urate of soda—such urate as constitutes gout elsewhere; these crystals generally occur in gouty cases, and are thought to signify gout, even when there is no history of gout or sign of it in other parts; but this conclusion is not so secure as it would be if urates were not passing constantly down the tubes.

When the microscope is used then we see that the change is of a simple kind. The cortical tissue of the kidney is wasted, especially under the depressions of the surface, the elevations or granules being the best part of the organ, though the name "granular disease" of the kidney seems to level the accusation at them. The wasting is seen to be very unequal, so that some bundles of tubes suffer more than others. They suffer in two ways, some shrivelling up, almost or quite completely, while others are dilated. The shrivelled ones often in parts are reduced to scarcely traceable cords, but in other spots a small portion of their epithelium is preserved, and this epithelium appears as a round or oval body composed of cells enclosing a hyaline-looking mass. These bodies were described as cysts by Mr. Simon, and were regarded by him as perhaps new structures, indicating a reconstructive effort to create new tubes; a similar view has been held regarding the relics of hepatic cells in cirrhosed liver. Other tubes dilate, and these are, perhaps, equally spoilt as to usefulness; the dilatation of tubes so tortuous forms cystic hollows, whose communications are hard to discover; these are the cysts described by Dr. Johnson. The epithelium in them is so thin as easily to escape notice. If we examine a portion of wasted kidney we may find some patches made up apparently of fibre, but in reality composed of wasted tubules; nevertheless, a closer examination shows good evidence of an interstitial formation of cell and fibre elements amongst the tubules. The tissue of the organ is never tough, as in cirrhosis of the liver. The Malpighian corpuscles are sometimes said to persist and be enlarged, and we have found many of them certainly larger than natural, but the important change in these corpuscles is a wasting of them with the wasting of the tubes they supply. In bad cases we have found one third of them utterly wasted to little thick-walled corpuscles, recognisable by their artery. The blood-vessels are often excessively affected, not only the main renal artery, but its branches, which we may perceive projecting on the cut surface, and which we often meet with in microscopic sections, showing very thick walls. This hypertrophy of the arteries is partly, no doubt, due to the resistance which the shrivelled Malpighian glomerules offer to the passage of the blood. But a general thickening of the arteries of the body is usually present in these cases, and they generally show more or less evidence of degeneration of tissue as well as thickening. It is sometimes said that the arteries are apt to be lardaceous in granular kidney, but this is only as a complication, for when they are so the arteries of the spleen, liver, &c., are at the same time affected.

The epithelium varies much in its state; it may be almost natural in places; it is often full of fat-grains, or granular; in the dilated tubes it is thinned out much, being spread like a pavement epithelium on their walls.

The term *chronic desquamative nephritis* rests on a supposition as to the origin of the disease of which there is no proof. Johnson, who makes all the diseases dependent on changes in the tubular epithelium, would see in the apparent increase of connective tissue only an atrophy of the parenchymatous structure, and there are those who on the supposition of a new growth of this material would see no difficulty in making it arise from the epithelium, as is said to be the case in the corresponding disease in the liver. As a matter of fact, in the small granular kidney all the tissues have undergone a change. With reference to the question whether the large white kidney and the small red granular one have the same pathology, or whether the one is essentially a parenchymatous disease and the other interstitial, it may be remarked that in the very few cases of chronic Bright's disease where there has been a history of acute disease preceding it, the latter has been associated with scarlatina; the form of acute nephritis which of late has been distinguished as glomerular. It has therefore been surmised that some forms of nephritis may terminate in the granular kidney, and especially that which follows scarlatina. No doubt a difficulty has been created by the kidney which Bright called mottled—a white kidney, somewhat larger than natural and granular on the surface. This might well be regarded as a large white kidney passing into the atrophic granular form. But the fact of acute inflammatory products being found in connection with chronic changes does not prove the inflammatory nature of the complaint any more than an acute pneumonia in a phthisical lung proves the disease to be pneumonic in its origin, rather does it show that an acute process may attack a chronically diseased organ. It should be said that those who believe all the forms of Bright's disease are but stages of the same nephritis would not necessarily maintain that the one passes into the other; that the red granular atrophied kidney had at one time been large and white, but that the initial inflammatory process had been the same and then taken two directions, the one towards the development of the larger form, and the other towards the slower and atrophic variety.

In chronic Bright's disease there is, as a rule, an alteration in the state of the heart and blood-vessels. The former is hypertrophied as regards the left ventricle, and the latter are thickened in their coats. This is seen to be the case by the naked eye in the renal artery, the vessel being open and rigid, the internal coat separating from the middle. In other vessels the character of the change has given rise to much controversy, the opinions which are held being as much opposed as is a physiological process to a pathological one, the former being a necessary and useful change, and the latter being a distinctly morbid one. Bright himself remarked this thickening of the coats of the vessels, and believed it was due to the irritating nature of the undepurated blood. George Johnson has laid much stress on this condition of vessels, and believes it is due to hypertrophy of the muscular coat brought about by the increased efforts which the small arteries and arterioles have to make in regulating the flow of blood through the organ. Gull and Sutton regard the change in the vessels as strictly pathological, that the thickened vessels which may be found throughout the body are composed of hyaline and fibrous tissue. The changes are strictly degenerative, and may be found in the smaller vessels of the brain, spinal cord, and other organs of the body. From this they propound a theory that chronic Bright's disease is not primarily an affection of the kidney but one of the vascular system. This they call *arterio-capillary fibrosis*, and regard the granular kidney as merely one result of the process.

We may say, in conclusion, that the general opinion in England at the present time is that the small atrophic kidney is a primary affection and does not originate by an acute inflammation as does the large white variety; moreover, that it is associated with disease of the blood-vessels, &c. At the same time, there is some evidence to show that an atrophied kidney may sometimes be found in those who have had a history of acute nephritis, especially if this be of a scarlatinal kind; that is, that it has originated in a glomerulitis rather than in an inflammation of the tubules. This form of atrophy is said also to be generally distinguishable from the ordinary chronic type, and is not associated with disease of the vessels.

It may be observed that chronic Bright's disease is often a concomitant of *gout*, and is also seen in those who have long been subject to the influence of *lead*. A typical specimen was presented to the Museum by Dr. Wilkin. It came from a girl who had never had any illness except severe lead poisoning. She died of uræmia. There also seems a tendency to the same change in the kidney of *old age*.

*Lardaceous kidney.*—Lardaceous disease is shared by other organs when present to any extent in the kidney; hence it is less properly a special kidney disease than the acute and chronic Bright's diseases we have described. Yet it leads to albuminuria, and, indeed, we have found indirect evidence that one at least of Bright's own figured examples of large white kidney was lardaceous, since the liver corresponding, which is preserved in the Museum, we have found to be highly lardaceous. The lardaceous change was, however, unknown to Bright.

The kidneys are at first enlarged and pale; the cortex smooth, its structure not confused or disarranged, but well defined; the pale cortex contrasts with the dark pyramids. The consistence at this early stage is little altered. If tincture of iodine be applied to a section, in a few moments dark points and streaks appear in the cortex or at the apices of the pyramids especially, these dark parts contrasting strongly with the yellow iodine stain around. The microscope shows that these dark flecks are Malpighian bodies, or minute arteries whose inner coats are seen to be swollen and glistening, and to have become of a deep walnut colour by the action of the iodine. If no iodine be used the arteries look tortuous and glistening, the peculiar refractile lustre of the lardaceous matter prevents the microscope defining the minuter parts affected. The renal tubes are widened and their epithelium is flatter and thinner than natural. Often in the channels of the tubes are hyaline casts, which we have never seen to take the colour of amyloid matter by iodine, though other observers describe it so. The epithelium is perfect around these casts. In this stage the urine is very free and pale; at first without, or with only occasional albumen; gradually, afterwards, with much albumen. The disease begins in the arterioles and tufts of the glomeruli, the walls becoming swollen and translucent, and afterwards the tubes are involved. When the disease is long established and advanced the organ is, perhaps, shrunken again to its natural size, or smaller, its outline being uneven, and the capsule thick, its state, indeed, approaching that of granular kidney. The microscope, too, shows wasting, &c., of the tubes as in granular kidney. Also degrees of catarrh may be noticed, and so the condition sometimes of large white kidney may be approached. Hence it is that some observers regard the lardaceous condition as only an accident supervening on Bright's disease. But against this view we shall see that the peculiar characters of the early stage are decisive. For a long time the lardaceous state is independent, and afterwards upon it come the catarrhs, &c., just as they may supervene upon granular kidney. So it is that we get mixed forms. The lardaceous change never progresses far in the kidneys without developing also in other organs. When this is the case the arteries and capillaries are much diseased, and the walls of the tubules in places affected; even the epithelium may be attacked, but this is more rare than is commonly described. The amount of lardaceous material now affects the consistence of the organ, which feels waxy, and looks dry and waxy on section, especially at the tops of the pyramids.

Lardaceous disease generally arises from syphilitic or strumous cachexia, especially when associated with suppuration. Any prolonged suppuration, as from old carbuncle or cancer, may cause it. Dr. Dickinson hence calls it "depurative" disease, deriving the name from his theory of its origin, which assumes that lardaceous matter is probably dealkalised fibrin, and that pus, which is alkaline, carries off alkali from the blood, so that prolonged suppuration at last leaves some fibrin dealkalised in the vessels. Such views may constitute a step towards clearing up the great obscurity surrounding the lardaceous change ; but it is not shown that by suppuration there is an absolute loss of alkali, and we meet some examples of very severe lardaceous disease where there has been no suppuration. This has been especially the case in syphilis.

**Hydronephrosis.**—In this condition the kidney is wasted and stretched about its pelvis, which is dilated through obstruction of the ureter. Thus a great cyst is formed, which may, from its size, be mistaken for a large ovarian tumour filling the abdomen, and an operation may be attempted for its removal under this belief. When in an extreme degree, the obstruction is commonly due to a calculus impacted in the ureter, or to a cicatrix of old disease arising from the passage of a calculus. But lower degrees are often bilateral, being due to obstruction in the urethra or bladder, or to pressure on the ureters by tumours, &c., from without. The stages of the distension are interesting. Naturally the pyramids fill the calyces, but when obstruction causes accumulation in and tension along the ureter, pelves, and calyces, the latter widen and the pyramids shrink in a curious way, the circumferential drag of the distended calyx attached around the root of each draws it down so that it becomes flattened out at its base, while the apex still for a long while appears as a little prominence ; at last this disappears, and in place of the pyramid a hollow is found, which hollow is continuous with the interior of the calyx. This occurs in each calyx until the kidney is spread out to form the walls of large cavities, the pelvis meantime dilating enormously, so that the wide calyces appear as sacs jutting from one side of it, the septa between the calyces being composed of the "columns of Bertini," which originally separated the pyramids from each other. Such hydronephrosis may suppurate through calculous or other irritation, and thus a large abscess or *pyonephrosis* form, causing adhesion of the contiguous viscera and tending to discharge itself in the lumbar region, or more rarely by the psoas or colon, &c.

**Cysts.**—Cysts arising in the proper substance of the kidney are very different to the hydronephrosis thus described. These are of three different kinds :—First, there are simple cysts of no great importance, frequently enough met with, especially in the bodies of elderly people ; there may be one or more, but only a few, of considerable size, projecting in relief from the surface and sometimes large, the rest of the kidney being healthy and quite adequate to its office. They arise in the cortex of the organ. Their contents are mostly fluid, and when analysed are found to contain a small quantity of albumen, and the ordinary salts existing in the fluids of the body, but very rarely any urinary ingredients. Very often these cysts contain a thick jelly-like matter, sometimes like thin glue, and at others nearly solid, which resembles somewhat the colloid matter found in other parts of the body.

A much rarer kind of renal cysts, but of greater clinical importance, because the disease proves fatal, is where the kidney is converted into a mass of cysts, as may be seen in several of our specimens, some of them holding several ounces of fluid; in these cases the renal structures may be reduced to mere relics found between the cysts; indeed, the septa between these are found to be composed of renal tissue which would not be expected by their appearance. This renal tissue is then in the state of granular disease; sometimes a remainder of the substance of the kidney in its proper form is preserved on one side of the organ. Some of these cystic masses of disease are ten times the normal size of a kidney, forming, indeed, a very remarkable pathological change. One pair of cystic kidneys weighed $8\frac{3}{4}$ lbs.

The clinical history is the same as that of chronic Bright's disease, of which, notwithstanding their vastly different appearance, these enormous looking tumours form only a variety. The cysts are, in short, an excessive production of that minuter cystic condition of the kidney which we have already described as occurring in granular kidneys. But there is another direction in which their pathology bears, which must be known. Of late years attention has been drawn, especially by Virchow, to a cystic change of the kidney, which is found in stillborn children, or as a congenital condition fatal very early. The kidney is found converted into a mass of cysts; this state often coexists with other malformations. Sutton thinks they may be formed from the tubules of the Wolffian body like the similar cysts in the testis and ovary. It is a natural question whether the great cystic kidney of adults represents a lower degree of the fœtal cystic kidney which has proved viable. In favour of this view is the discovery of such cystic changes sometimes in young subjects, although the majority of them occur about early middle life. We have already suggested reasons for believing that some examples of small granular kidney represent originally ill-formed organs.

The last kind of cysts are those found in immense numbers and of microscopic smallness, which we have already fully described when speaking of granular kidney, to which they properly belong, and, indeed, of which they form one of the most characteristic features.

**Fatty Kidney.**—When, a few years ago, the microscope was put into use to discover the nature of Bright's disease of the kidney, and fat was found in the tissue, an opinion was ventured that the disease was a fatty degeneration of the organ. To test the correctness of this, some diseased kidneys were submitted to chemical analysis, and found to contain no fat, when the opposite statement was made, that Bright's disease is not a fatty change. Both statements are true, but their application is, however, limited; the large white kidney of acute

Bright's disease often contains a large quantity of fat, while the granular kidney of chronic Bright's disease may contain little or none. In the first case, however, the presence of fat is altogether a secondary affair, and due to a change or degeneration of the inflammatory products, and therefore the term fatty, as applied to the essential nature of the disease, must be as erroneous as it would be to call a cancerous tumour or a phthisical lung fatty because both contain an abundance of oil. We think, therefore, the term fatty kidney should be only used in the same way as we use the term fatty liver, *i. e.* not for all morbid conditions where fat is found, but for those instances where the fat constitutes an independent diseased condition. Such a state is often met with in cases where the other organs are fatty ; a small quantity of fat is generally present in ordinary kidneys, but in those we are speaking of the epithelium is loaded with it so as to be quite opaque, in microscopic sections, and by reflected light to look creamy and opaque, but not very white, because there is no inflammatory accumulation, and hence no expansion of the tubes compressing the vessels. The function of the kidney in these cases may be disturbed, but as in the similar example of fatty liver, the surprise is that with so striking a change in microscopic appearance so little vital disorder is evinced. Certainly there is no albuminuria or Bright's disease in these cases. As to the causes of this fatness of the kidney it occurs often in the large kidney of diabetes; in this it seems to correspond with the like fatness of hypertrophied hearts, which appears to represent the failure of nourishment of the overgrown elements before dissolution. It is also found in the general fatness or "steatosis" of phosphorus poisoning, and to a less degree in chronic antimonial and arsenical poisoning. It also occurs in some cases of great obesity, and sometimes we could not account for it. It must be clearly distinguished from Bright's disease.

**Fibrinous Deposits or Embolic Infarctions.**—These are of the same character, and have the same origin, as those in the spleen. We said, in that association, that such deposits are found in cases of heart disease, especially in those where the mitral valve is affected and its curtains covered with vegetations, whence it has been thought that some fragment from the latter, being taken into the circulation, is not arrested until it arrives at the minute capillaries of a parenchymatous organ ; that there it lodges, obstructing the blood-vessels and producing a change known as capillary phlebitis. The method of production, however, is not yet universally agreed upon ; all observers allow that embolism will produce these infarctions, and by all it is held as certain that an obstruction occurs in these parts, and that the vessels are blocked up, so that, owing to the impediment in the circulation, a stagnation of blood takes place in the affected part. In consequence of this the blood and the tissue undergo degeneration together, so that the red colour is lost from the centre outwards, while, perhaps, additional changes arise from inflammation. Thus, in the kidney, as in the spleen, a pale mass is seen surrounded by a dark red halo; the mass is wedge-shaped, owing to the mode of distribution of the blood-vessels, which correspond to the wedge-shaped radial segments of the kidney; the base of the wedge is outwards, and slightly protruding; the colour is at first of a dull purple, afterwards becoming white, commencing in the middle. If examined by the microscope the structure of the affected part is still seen to persist, but there is a little granular or fibrillated material between the tubules, and these gradually lose their definition of outline. After a time the part begins to decay, it is more granular, and the tubes themselves are confused with granules. In an early volume of our 'Reports' it is figured by Bright and Barlow. After a time the mass shrinks until it quite disappears, leaving a cicatrix; and thus in the same kidney we may find instances of the affection in various stages—some masses wasting, leaving a depression on the surface; others not having altogether disappeared, and others which have left a deep cicatrix; we then see that the tissue amongst which they form becomes at last destroyed. Sometimes, though not commonly, they soften into a purulent fluid; when this is the case the embolus is infective and generally arises from ulcerative endocarditis as has already been described.

A plugging of the whole renal artery has occasionally occurred, with very different results in different cases. Cohnheim gives a case where he considered the effect to be a necrosis of the organ. Dr. Charlewood Turner has found in many cases the papillæ and portions of the pyramids necrotic and partly detached. This has been due to a thrombotic obstruction of the arterioles or defect in the general nutrition. He has regarded the necrotic portion of the pyramids as analogous to the parts in the cortex which had undergone absorption, the difference in the process due probably to the urinary secretion. We met with a case of obstructed artery where Bright's disease of both kidneys was already present, and an example recorded by Mr. Pick showed granular disease of the kidney as the result of such obstruction. In our case the cause of the arterial obstruction was embolism with an ante-mortem clot from the left ventricle.

**Tuberculous Disease.**—This is of two kinds: one, where miliary tubercle is scattered throughout the kidneys as well as in other parts of the body in general tuberculosis; this is unimportant clinically, but offers a good opportunity of studying the origin of miliary tubercles. We see crowds of lymphoid corpuscles arising in the interstitial tissue of the affected spots; these spots are always small, and caseous change very quickly appears at the centre of the tubercle. The other form, which might be rather called a *scrofulous inflammation*, constitutes a primary and fatal disease. It is sometimes called strumous pyelitis, since the pelvis is soon affected. The leading symptom is a large amount of pus in the urine. After the continuance of the disease for some years the organ becomes quite destroyed; large hollow spaces are formed in it with only a trace of the secreting tissue between them; the mucous membrane is thickened, covered with scrofulous matter, and beneath the membrane there is the same matter running into the tissue. The first stage of the disease is met with now and then accidentally; we find a caseous patch perhaps softened; it is situate just under, or at the surface of, one of the cones, or several cones may be affected. In other cases we find the preference is for the pelvis of the kidney, so that this is scattered over with tuberculous ulcers, the cones being little affected, or perhaps not at all.

These two modes of origin lead to two distinct developments in the more advanced stage. For in those cases where the pelvic mucous membrane is most affected there generally soon occurs some obstruction of the entrance of the ureter by inflammatory swelling or by an ulcer there. This causes a dilatation of the pelvis and calyces, &c., like the dilatation which produces the "hydronephrosis" we have recently described. The remains of kidney tissue are then compressed, the pyramids flattened down, and calyces expanded, so that an enlarged and sacculated cyst, full of pus and lined with a tubercular suppurating tissue, is all that remains. We once met with such a cyst having a quantity of remarkably large and bright flakes of cholesterine in its contents. This is very rare; there are but few instances of it on record. In the other form the renal tissue suffers most severely, and although the pyramids, we believe, are always earliest and most severely affected, yet we may find in the thickness of the cortical part of the organ large vomicæ or caseous abscesses, which run together and increase and multiply till the whole organ is destroyed. We see by our specimens what a great destruction of the organ occurs. Sometimes we meet accidentally with examples where such a disease has been cured; of course the organ is destroyed, but the ureter has become closed, and the purulent and scrofulous matter, having been encysted, is now converted into a putty-like mass; when we meet with such masses accidentally in inspecting a body, we may regard them generally as dried-up abscesses. But sometimes the abscesses burst externally in the loin, or open into the colon. The disease is often unilateral, but after a long time the other kidney is apt to be affected. Sometimes we see miliary tubercles in the renal tissue around the scrofulous ulcers and abscesses in the pyramids. It appears that this disease is a primitive tuberculosis of the kidney, and so represents the primary renal phthisis; miliary tubercle, on the other hand, is always secondary. We have seen the primary scrofulous kidney set up general tuberculosis, just as pulmonary phthisis too often does. Its extension down the ureter to the bladder we shall presently consider.

**Carcinoma.**—As in general tuberculosis, so in general carcinomatous disease, we may find deposits scattered through the kidneys as well as other organs; but it is only after death that these are discovered. In this way sarcoma or melanosis or colloid of the kidney may be met with, as well as carcinoma. Where the kidney is the ordinary seat of cancer there is usually a tumour, and the disease is recognised during life by the symptoms it produces. Yet in these cases it is remarkable how seldom the proper tissue of the kidney can be said to be primarily affected, for although a large tumour may exist, and this after death be removed as renal cancer, yet a careful dissection discovers the disease to have arisen primarily in the lymphatic glands, or other parts outside the kidney, especially about its hilus, while the organ itself is comparatively untouched ; the disease, however, puts on the form of the kidney, for it has probably penetrated the hilus and expanded the capsule ; this, we think, is the commonest plan of attack in primary renal cancer ; it may be long before the pelvis of the organ is penetrated, and, as the proper tissue is nearly intact, we can understand the frequent absence of diseased products in the urine. Occasionally, however, the whole structure may be infiltrated and immensely enlarged, the kidney being converted into a large white mass. In the same way as biliary calculi and cancer are associated, so we may have renal calculi and cancer together, as displayed in one of our drawings.

Occasionally, however, cancer is seen to be a primary affection, and Moore has shown it originating in the epithelium of the renal tubules, and Sharkey in the Malpighian capsule. A remarkable specimen is on our shelves consisting of a very soft growth which appears to be cancer, and containing a quantity of fat. This is probably the *lipomatous cancer* of Cornil and Ranvier.

*Epithelioma* of the kidney as a secondary deposit to similar disease elsewhere, we have more than once met with. There were well-marked capsulated bodies and other characteristic elements of the disease, whilst remains of the glomeruli and tubules were still seen within the white masses which were scattered through the organ. In one case the masses were very large and softening, so that fluid matter like pus ran out when the capsule was removed. The epithelial nature was well marked and exactly resembled the deposits elsewhere.

A *congenital sarcoma* occurring in infants has been met with several times ; a large white tumour fills the abdomen, which on examination is seen to be made up of spindle-cells. It has been thought that these have originated in embryonic structures connected with the Wolffian body, owing to the peculiarity of their formation, for Dawson Williams discovered in a case of this kind striped fibre which exactly resembled muscular fibre, and Mr. Eve had a similar case where he found scattered amongst spindle-shaped cells some interlacing ones of striped muscular fibre. These congenital growths, therefore, have been designated *myo-sarcoma.* Dr. Paul examined a tumour of the kind and he found in it rows of cells resembling groups of tubules of the fœtal kidney ; in fact, an adenoid structure, and this he therefore styled an *adenoid sarcoma.* Besides these congenital diseases of the kidney there is the cystic disease already spoken of.

In Hodgkin's disease, or *lymphadenoma*, white deposits may be met with in the kidney as in other organs. They are mainly composed of masses of small cells. In cases also of enlarged spleen and liver known as *leukœmia*, small groups of white cells may be found.

*Cavernous angioma* is much more rare in the kidney than in the liver, but is occasionally met with.

*Syphiloma*, or syphilitic gumma, we have once met with, but it is rare. Very few cases are on record.

*Villous* disease of the pelvis of the kidney, similar to that in the bladder, has been very rarely met with; in a case by Dr. Murchison there was villous disease both of the kidney and bladder.

*Pigmentation.*—The kidney may be pigmented like other organs in intermittent fever. Grey spots are seen on the cortex and dark lines on the pyramids, showing that the pigment is in the capillaries and Malpighian bodies. Our shelves contain a portion of kidney pigmented in a somewhat similar manner from nitrate of silver.

**Parasites.**—*Hydatid* disease of the kidney is not very common. We often have patients before us with renal tumours in whom it is suspected, and we occasionally hear of cases where hydatids pass in the urine; but in the post-mortem room they are not often met with; in two or three instances we have found them accidentally where no symptoms were noticed during life. They were on the surface of the organ, and had not interfered at all with its structure; in one of them the kidney was expanded around the cyst, but apparently quite healthy. In one case there was a hydatid in the liver, in the others the disease existed alone. In all of them echinococci were found.

*Cysticerci* have occasionally been found in the kidneys.

*Bilharzia* is a kind of fluke infecting the kidney in South Africa and causing hæmaturia. It has already been mentioned under "Intestine." The ova, which have a sharp point at one end, may be found in the urine. When warmed they soon hatch, and the embryos escape showing a head and narrow neck.

We would here also mention the escape in the urine of certain minute entozoa from the blood. Worms were long ago noticed in "chylous" urine; but it has been shown by Dr. T. R. Lewis that these worms, which are named *Filaria sanguinis hominis*, are present in enormous numbers in the blood in these cases. They have already been mentioned under "Lymphatics."

**Calculi.**—It is not unusual to meet with calculi in the kidney after death. In some cases both organs are found filled with them, the structure has been destroyed, and they have become moulded to the form of the pelvis and dilated infundibula; thus we see large knotted and branching calculi fitting into the hollows of the kidney. They may be formed of uric acid, phosphate, and carbonate of lime, oxalate of lime, &c., and sometimes very beautiful semitranslucent white calculi are composed of cystic oxide. Occasionally they cause great changes in the kidney itself in the form of grave pyelitis and pyelonephritis, with the usual characters of these states before mentioned; or by inducing ulcerative or other inflammatory contraction of the ureter in passing down it, or else, by lodging in and obstructing it, they lead to dilatation of the pelvis of the kidney, which may be combined with inflammation, and in this way an enormous cyst containing pus may arise, which has been known to rupture into the abdominal cavity; or, this being prevented by adhesions to viscera around, the abscess has been evacuated by operation, or in any of the natural ways before mentioned in speaking of pyelonephritis. At other times we find that a kidney whose ureter is totally obstructed by a calculus has dwindled away to a small relic; whether this happens without any previous dilatation we cannot say.

## URETER

**Malformation.**—One of the most common diviations from the natural condition is the presence of two ureters to one kidney, and this sometimes is associated with a double pelvis to the kidney; these two ureters may pass separately to the bladder, or unite before they enter. We have several times seen these variations.

**Dilatation.**—This is a condition very frequently met with, owing to an impediment to the flow of water to the bladder. Thus in stone, and various chronic diseases of the urinary passages, it is found; also from direct pressure on the ureters, as from ovarian disease, cancer of the uterus, &c. The dilatation is generally not uniform, but unequal, producing a sacculated appearance. The ureters are sometimes distended to the size of the intestine; numerous examples of this can be seen on the shelves. When inflammation complicates the condition the ureter is thickened as well as dilated.

**Obstruction.**—This is often associated with dilatation, of which it is the cause; thus a twisting of the ureter, or stricture from previous ulceration, or else the presence of a calculus, will all produce these conditions. We have specimens showing calculi in various parts of the canal. Some cases appear to indicate that a sharp blow on the abdomen struck over the ureter may cause inflammation enough to lead to subsequent obstruction.

**Inflammation.**—We have already said that in cases of suppuration of the kidney the disease may have extended upwards from the bladder, so that not only the latter organ but the ureter is found to have its mucous membrane covered with adherent flakes of lymph, and pouring out a muco-purulent secretion. In some cases the coats are infiltrated, and consequently thickened. The signs of inflammation are generally less in the ureter than in the bladder or kidney.

**Tuberculous Disease.**—This is generally associated with similar disease of the kidney and bladder, as we shall see by examining the preparations of these organs. The internal surface is covered with a scrofulous matter, the mucous membrane having long been destroyed, the walls are often extremely thickened, so that on section they look like a solid stem with a small canal running through its middle. This narrow channel can be easily closed by any fresh local swelling in its wall; and such an occurrence we shall find indicated frequently in the clinical course of many of these cases, by the occasional augmented pain with reduction of the pus in the urine, such periods of greater pain being terminated by relief, coexistent with the reappearance of the free flow of pus.

**Cancer.**—In the same way cancer is not a primary disease of the ureter, but only occurs in connection with cancer of the bladder when the adventitious growth sometimes proceeds upwards in the course of this tube.

**Injury.**—The ureter generally escapes, except in cases of excessive violence in which other fatal damage makes its condition unimportant; but sometimes gunshot wounds will specially select the ureter, as in the case of an Archbishop of Paris. Very rarely, also, in a crushing accident, the force has by chance fallen destructively on the ureter, causing extravasation of urine in the lumbar region; we have seen two such instances. We have mentioned the occasional obstruction of the canal as a consequence of milder degrees of such injury. On the subject of injuries to the ureter there may be read with advantage an able paper by Mr. Poland in the 'Guy's Reports.'

## BLADDER

**Malformation.**—The most common is extroversion of the bladder, accompanied by malformation of the urethra; a fissure exists in the abdominal walls, the bladder is exposed, and the ureters are seen opening directly upon it, as may be observed in our various specimens. The malformation generally extends up to and includes the umbilicus, and below it reaches and includes the genitalia, which are rudimentary and malformed, the pubic arch being incomplete. But in some cases the malformation more especially affects the bladder, the umbilicus above being pretty well formed, and the pubes and genitalia complete, so that the bladder forms a more or less protruding red pouch. The exposure of the bladder in these cases gives us an opportunity of examining the urine direct from the kidney, so that if we place a piece of test-paper to the opening of the ureter, we find that the urine is strongly acid as it is poured into the bladder.

**Hypertrophy.**—This is a very common result of impediment in the urinary passages, and arises from stricture, enlarged prostate, &c. The muscular wall becomes much thickened, and on the inner surface of the organ the muscular bands are seen as strong cords interlacing with each other, not unlike the columnæ carneæ at the apex of the hypertrophied right ventricle. Whenever we find the bladder thus thick and reticulated within we should examine the urethra for stricture; low degrees of the condition are common enough; we often see a distinct but slight hypertrophy caused through the obstruction of the urethra in dropsy of the prepuce. If the hypertrophy of the bladder is efficient there may be no *dilatation;* this, however, very often accompanies hypertrophy, so that the organ becomes much enlarged in size as well as thick. Mr. Fenwick says that in hypertrophy the anterior wall is always thicker than the posterior.

But in hypertrophied bladders it is far more common to find the organ *sacculated* than uniformly dilated. Sacculation of the bladder is a consequence of the distribution of its muscular tissue; this is not uniform, but the fibres are disposed so as to make a meshwork of strong bundles whose intervals are filled in with irregular weaker bundles. Under the greater pressure of the hypertrophied bladder when resisted by stricture, &c., the weaker meshes yield and the mucous membrane is forced out, at first but little, so that stretching of the bladder will efface the sacs; but afterwards they protrude outside the muscular layer, through its meshes, and reach and raise the subperitoneal tissue; thus numerous pouches may be formed chiefly in the sides and upper part. The sacs are composed of mucous membrane covered by peritoneum, the submucous and subperitoneal tissues blended between them. Sometimes there is one large saccular dilatation, equalling the bladder in size; we met with one curious case where the bladder was severely inflamed, while a sac from its side, of greater size than the bladder itself, was quite free from the inflammatory action; upon these larger sacs we may sometimes see a few muscular bands running in their walls. Cases are recorded where great distension of bladder has been found in the fœtus, from some obstruction in the passage below. Sacculated bladders have been found congenital. These sacculi may contain calculi, which then are said to be "*encysted.*"

*Simple or acute dilatation* of the bladder is of great importance. It is found in cases of fever or of injury to, or disease of, the spinal cord or brain, &c., conditions where the organ is paralysed and the urine retained. If this dilatation is not attended to it may lead to disastrous consequences in sloughing or, perhaps, rupture of the viscus.

**Atrophy.**—This is especially seen in long-standing cases of paralysis of the sphincter of the bladder, where the organ is never distended, but empties itself as soon as it receives any water from the ureter. Thus in cases of recovery from spinal paraplegia, where yet a constant dribbling of the urine is present, the bladder has permanently contracted, and become, as it were, merely a part of the channel between the kidney and urethra.

**Injury.**—The bladder is frequently cut in the operation of lithotomy; when we have met with such cases years afterwards we have been unable to discover the slightest trace of the wound. It is also sometimes punctured through the rectum, to evacuate its contents in cases of stricture. Besides more recent cases, three examples of some duration have come before us on the post-mortem table: in one, where death occurred a few months after the operation, the opening still remained between the two organs; in the second, where death occurred a year afterwards, the fistulous opening was with some difficulty found, it had not allowed any urine through it, except for a short time after its production; in the third case a small depression or hole existed just behind the prostate, but it was blind, and the wound in the rectum was perfectly healed, so that not a trace of it could be found.

The bladder may also be wounded by the point of the catheter. This is a very serious form of well-intentioned injury. In cases of cystitis after retention and catheterism one should always look well at that part of the back of the bladder which is opposite the urethra, and upon which the point of a catheter would impinge when introduced too far; we may then often see the cause of the cystitis. Injury to the bladder through accident occurs generally in cases of fractured pelvis, either from a heavy weight falling on the body, or a cart passing over it; the bodies of the pubes and ischia break, and the sharp ends may pierce the bladder; or the bladder may be pierced by weapons, producing fatal extravasation, though a few cases recover, with urinary fistula. When the abdomen has been opened and the wound closed, a perfect recovery has occurred.

Sometimes, as we have more than once seen, the bladder has been ruptured by similar causes, without any fracture, when blood and urine are poured into the abdomen, setting up fatal peritonitis; the bladder, when full, has also been burst by direct violence, as we have seen, from a kick in the abdomen; and there is reason to believe it has happened in the female during coitus. The most interesting question in reference to rupture of the bladder is, whether this can occur spontaneously from excessive distension; such cases are related, and we have a specimen supposed to represent a rupture from such a cause; but it shows also, what we have always met with ourselves, a previously diseased condition of the organ. In the few such cases which have come under our own notice the history has been one of long-standing stricture, and subsequently of disease of the bladder; and after death a general and local peritonitis has been found connected with a sloughing condition of the latter organ, allowing the urine to escape through the coats. In these cases there was always a history of previous disease, which was sufficiently evident by post-mortem appearances; we have never yet seen a case of spontaneous rupture of a healthy bladder.

**Inflammation or Cystitis.**—We have already had some occasion to remark against the opinion, which was formerly held too exclusively, that acute inflammations are the primary and typical diseases to which the body is liable; for, in fact, most of the changes in the body are chronic, and from these the acute diseases result secondarily. The term cystitis suggests these remarks, for, common as it is in connection with stricture, diseased prostate, calculus and other chronic affections, it is very rare, if indeed it ever occurs, as an idiopathic acute disease. A calculus may set up cystitis and then itself escape per urethram. This has been held to explain some cases of apparently idiopathic inflammation associated with dilatation of an ureter. It is remarkable, considering the frequency with which the bladder is allowed to become over-distended, that severe inflammation from this cause is so comparatively unfrequent.

But cystitis caused by retention of urine or by foreign bodies will be found frequently enough; thus in persons who die with calculus in the bladder, or stricture, various degrees of *chronic inflammation* are constantly met with; the mucous membrane is sometimes destroyed in parts by ulceration, but more generally it is altered in character, thickened and congested, so as to be like red or purple velvet, or in severe cases it is covered all over or in patches with granular lymph, when the state receives the name of diphtheritic or pellicular inflammation. But we must not mistake for this the concrete layer of adherent mucus often mixed with phosphatic secretion, which is commonly found in the inflamed bladder. The graver changes are found at first on the summits of the rugæ, and always most advanced there. This appears to arise partly because the rugæ are kept exposed to the urine in all states of the bladder, whilst the recesses between them are closed by the coaptation of their sides; partly, also, it is due to the congestion which the summits of the rugæ suffer in comparison with the sides, which are kept more empty of blood by their coaptation and mutual pressure. The urine is purulent and alkaline. It is found to contain some peculiar micrococci from which fresh urine is quite free. The most severe and *acute inflammation* of the bladder is generally met with in those cases where the organ has become paralysed from injury or disease of the spine, and especially if catheterism have been much adopted. In these cases the bladder becomes distended, the urine ammoniacal, the surface of the bladder inflamed all over, or, as we have already hinted, especially where the point of the catheter has touched it, it may have a slough at this spot. We have a specimen from a case where death occurred a week after the first symptoms, and where the whole mucous membrane is detached, and hangs loose in the bladder. Cases are recorded where more or less of, or even all, the mucous membrane has been passed through the urethra. It is a question much discussed whether this intense inflammation and tendency to sloughing of the bladder (as well as the bedsore, which so often occurs in the same class of cases) is due to the nervous influence being removed from the part; and in this way acquires its proneness to inflame, as some believe the cornea does when the fifth nerve is affected; or whether the inflammation be rather due secondarily to the paralysis, from this causing a retention and subsequent decomposition of the urine, which hence acquires irritant properties and tends to inflame the bladder.

*Pericystitis* is a term which we use for inflammatory processes around the bladder, in the subperitoneal tissue, and is not uncommon as a consequence of many pelvic diseases. The most acute form is seen from infiltration of urine, beginning at the neck of the bladder, and then pervading all the structures, so that when, at the inspection, the peritoneal coat is cut through, the cellular tissue beneath is found full of purulent matter, or even sloughing. Such suppuration may spread to the sheath of the rectus and other parts of the abdominal wall, and so require external opening. Thus sometimes from lithotomy such infiltration has occurred, producing this grave condition, and lighting up at last a general peritonitis. The same thing has been known to occur from pressure of a calculus ulcerating its way through the bladder. In cases of stricture, too, where the urethra is much diseased, so as to be perforated by ulceration or by the catheter, a like escape of urine may occur, with all the grave consequences we have enumerated, although then the perforation is in the urethra; we have examined a great many cases of fatal stricture and extravasation of urine, and, besides the subcutaneous inflammation, we have very often met with a similar inflammation, suppuration, or sloughing in the cellular tissue of the pelvis, behind the deep triangular ligament. This has been due to the mischief about the stricture proceeding backwards to the neck of the bladder, and involving the prostate in the inflammatory process, whence the disease extended around the bladder.

Instances have occurred where an abscess, arising from the presence of a calculus or other cause, has caused a fistulous opening into the rectum, or in the abdominal walls above the pubes.

In one specimen we see extravasation of blood beneath the mucous membrane, by which it is raised up into a number of projections resembling the surface of a placenta.

**Tuberculous Disease.**—This occasionally occurs as a primary disease in scrofulous subjects, but far more frequently it exists in connection with chronic tuberculous disease of the kidney. The latter organ is generally found in a state of more advanced disorganisation, having been first attacked by the disease; the ureter is next involved in the process, which thus spreads down to the bladder. This is found thickened, its inner surface showing tuberculous ulcers, as seen in our preparations. The ulcers arise by the softening of miliary tubercles in the mucous membrane, just as in the bowel; hence they are at first small and round, but other tubercles form and spread and open in the floor and margins of the ulcer, so that it increases and becomes complex and deep with a thickened base. By the spread of this process the mucous membrane may be almost entirely removed, while the muscular coat is infiltrated with the tubercles. We sometimes meet with it as an extension upwards from the bladder.

**Morbid Growths.**—The bladder may be involved in cancer of neighbouring parts; this is especially frequent in cancer of the uterus. The back of the bladder may be laid open by this into the vagina. The spread of cancer from the uterus is generally continuous through the coats of the organ, but we have seen nodules of cancer appear in the cystic mucous membrane when there was no continuity of this with the uterine cancer. We have seen the bladder invaded by cancer of the vesiculæ seminales.

*Carcinoma* in any of its forms may occur in the bladder, but a primary scirrhus is rare, whilst *epithelioma* is more common; it shows its usual characters in spreading locally and affecting the neighbouring glands, but not reaching remote parts. In one case there were secondary deposits throughout the peritoneum, and in another the fungating mass in the interior was associated with growths in the bladder walls, and these again united to cancerous deposits outside in the peritoneum. The mode of spreading of cancer in the wall of the bladder varies; sometimes it extends as an equable thickening, as in one specimen; at other times it forms a local mass that may project in a polypoid form. The surface of a cancer within the bladder, like that of some cancers in the mucous membranes generally, may grow into villosities. Thus, in one specimen, where a thick mass was growing in the mucous membrane of the bladder, the surface was shaggy; and on placing it under the microscope, it presented the appearance seen in the drawing, exactly resembling the villi of the chorion. Yet, as we shall presently mention, most villous tumours are not of a cancerous nature. Virchow has described a tumour with a stroma of smooth muscular fibre and nests of cancer-cells in the meshes under the name of *myo-carcinoma.* As in other parts cancer is often set up by an irritant. In one of our specimens it is seen growing around an old fistulous opening, and in another it is seen in a bladder long irritated by a calculus and which had been operated upon.

*Villous growth.*—This is a very remarkable kind of disease; it occurs chiefly in young persons; small tufts grow on the mucous membrane, and cause death by hæmorrhage. We have now seen several of these cases; among others a railway porter, young and strong, was seized with hæmaturia, and in the course of a few weeks, in spite of all treatment, died from loss of blood. All that was found in the body were two small tufts scarcely larger than peas, and from these the fatal hæmorrhage had occurred. They appeared like little tufts of moss growing from the mucous membrane; and when examined by the microscope presented villous processes, and what is very striking, as may be seen in our drawings which we made at the time, the surface is covered by columnar epithelium, long battledore-shaped nucleated cells, very different from the ordinary epithelium of the bladder. Each villus, we should have said, contained loops of blood-vessels. Another example was from the body of a woman; the whole patch was not larger than a shilling; the hæmorrhage was very profuse.

*Polypoid* tumours not infrequently occur in the bladder; some have proved to be vascular, consisting of a deep-coloured mass of blood-vessels with but little to connect them. Others were of a fibrous texture (*fibroma polyposum*), others have more a *fibro-sarcomatous* texture. Small *fibrous* tumours form occasionally in the submucous tissue, especially near the neck; some of these partake of the muscular structure of the prostate gland and so are *myo-fibromata*, or myo-sarcomata. These polypi give rise to a fatal disease of the bladder, not, however, generally by hæmorrhage, as in the preceding case, but by interfering with the flow of urine, causing retention, cystitis, and consequent suppuration of the kidney. We had an opportunity of seeing two of these cases lately, and they both occurred in children. So also in the specimens in our Museum, and in other cases of which we have read, the subjects have been children. We shall see by these how the polypi grow within the bladder, and in considerable number, having peduncles, and being of a pyriform shape; their texture is soft, and composed of fibro-sarcoma; some of the smaller ones have a warty character on the surface.

**Foreign Bodies.**—Our Museum, besides showing calculi, has various other substances which have been removed from the bladder, both male and female:—Pieces of catheter, slate-pencil, bodkin-cases, knitting-needles, &c., and these, it will be seen, are covered with phosphates. Also a bladder containing the remains of an extra-uterine fœtation.

**Prolapsus** of the bladder sometimes occurs in women during labour.

*Dermoid cyst.*—A bottle in the Museum contains two pints of fat drawn off by a cannula through the rectum, from a young man who had a pelvic tumour pressing on the bladder. Ten years afterwards he was in the London Hospital for calculus. This when removed was found to contain a hair for a nucleus.

**Hydatids.**—We have specimens of hydatids passed in the urine, but they probably came from the kidney; and one specimen shows a hydatid growing outside the bladder.

*Bilharzia hæmatobia*, a minute fluke-worm, is found in the blood-vessels of the bladder, as a cause of hæmaturia, in Egypt and at the Cape of Good Hope. The ova have been found in the mucous membrane of the bladder as well as that of the large intestine.

## URETHRA

Although the urethra is organised physiologically more as a genital than a urinary apparatus, and its diseases are caused generally in subserving its genital functions, yet pathologically the effects of these diseases tell more upon the urinary system, since, even if they interfere with the genital process, they do not so produce those serious consequences which they entail when they impede micturition; they then often prove fatal. It is for this reason that we place the morbid anatomy of the urethra with that of the bladder and kidneys.

**Malformations** of the urethra concern more the generative function.

**Injury.**—This generally arises from a fall on the perinæum, which directly crushes the canal, or from fracture of the pelvis, in which the urethra is, perhaps, lacerated by a piece of splintered bone. But the frequency, nay, almost constancy, of rupture of the urethra in cases of fracture of the pelvis, shows that it cannot arise in this way. And an explanation of its frequency is found in the close mutual connection between the bones and the urethra by means of the triangular ligament, so that by the disruptive drag upon this ligament, in which the urethra forms, indeed, the weakest spot, the urethra is torn when the pubic arch is broken.

**Inflammation.**—We may sometimes meet with a simple *catarrhal* inflammation of the urethra in boys, also in *gouty* persons. But urethral inflammation is mostly due to *gonorrhœa*, and its specific nature is said to depend upon a micrococcus. In this disease the inflammation commences at first in the anterior half of the channel and gradually passes more deeply until it may reach the region about the bulb, where it lingers long. It is said the endoscope shows that the persistence of inflammation in this part is due to a granular state of the mucous membrane, like that observed in granular ophthalmia or granular metritis. The endoscope affords great advantages over morbid anatomy in detecting the characters of these inflammations, for such characters nearly disappear after death, when the vital turgescence has subsided. Desormeaux describes an herpetic kind of urethritis known by its patchy distribution.

In the few cases we have seen post mortem the mucous membrane was deep purple in colour from congestion; the diseased appearance extended throughout the passage nearly to the bladder, and was strongly marked at the perinæal bend of the channel; pus-like secretion was present on the surface. In mild cases only the mucous membrane is affected, and that merely with catarrhal inflammation, but the inflammation may become very intense in the membrane itself, so as to cause a pellicular exudation on its surface, or a formation of pus in its substance, causing it to have a whitish appearance. Or the inflammation may extend to the fibrous tissue beneath or to the corpus spongiosum, or even the corpus cavernosum. In two grave cases we have seen phlebitis of the prostatic plexus and pyæmia set up by this deep extension of gonorrhœa. Or the inflammation may spread to the bladder, or pass through the prostate and spermatic duct to the testes, causing inflammation in all these parts. This latter extension, however, belongs generally to the later declining stages of the disease, when the inflammation has reached the membranous urethra and is becoming chronic and granular.

A *plastic exudation* of lymph is not uncommonly met with in cases where there has been any grave injury to the canal, either from accident or in connection with operations. We more frequently find this in the post-mortem room in cases of old stricture, when there has been a necessity for laying open the urethra from the perinæum; here, perhaps from the incision, or more probably from the inflammatory process in connection with an extravasation of urine which has given rise to the necessity of the operation, we find the whole of the urethra, especially that part near the seat of the old disease, covered with flakes of lymph closely adherent to the mucous membrane, and sometimes forming a cast of the tube, or, instead of flakes of lymph on the surface, there may be pus in the substance of the mucous membrane.

**Stricture.**—Considerable obstruction to the passage of urine may arise through the changes in the urethra in acute urethritis, but stricture is essentially a chronic disease said to follow after a long course of granular urethritis; it is caused by a slow indurative inflammation of the submucous tissue, which hardens in becoming fibrous, and so constricts the urethral canal. We sometimes find this external induration extend into the cellular tissue all around, producing a hard tissue as dense as cartilage, and this is especially the case if connected with perinæal fistulæ; sometimes the part closed is very short, so as to resemble a fold drawn across the channel, sometimes prominences project into it, sometimes a portion of the tube is simply shut up. About perinæal fistulæ granulations may project into the urethra. It was formerly said that the usual seat of stricture was the membranous part of the urethra; but it was pointed out, we believe by Mr. Syme, many years ago, that the beginning of the spongy portion is its most ordinary situation. This is the most dependent part of the channel, and to this part inflammatory products would gravitate. Endoscopic examination proves that long after a gonorrhœa a granular inflammation lingers here. This part, also, is that which is most exposed to vertical pressure by the weight of the body in accidental falls upon the perinæum. From examination of many cases of stricture we can say that it occurs either at the first part of the spongy portion or at the point of junction of the membranous and spongy parts. We never find strictures in the prostate, but they are sometimes seated towards the glans.

Our shelves contain several specimens of stricture; in these may be seen false passages passing about the sides of the urethra in various directions. In some cases there are many; and in one example we found six distinct ways into the bladder. Sometimes these false passages are lined by so smooth a mucous membrane that it is hard to say which is the proper urethra out of so many. One specimen shows the urethra perfectly closed, the canal being quite obliterated.

**Vascular Growths.**—These occur mostly in the female, and are situated at the orifice of the urethra, forming a fringe around it. They are highly vascular and sensitive tufts, the villi containing loops of blood-vessels.

**Carunculæ** are spoken of as occasionally occurring in the male urethra, as small vascular growths on the mucous membrane, causing obstruction to the flow of urine. We have never yet met with them in the recent subject, although we have seen one or two so designated in museums.

**Cancer.**—The urethra may be involved secondarily in cancerous growths contiguous to it; thus we have seen a medullary carcinoma of the bladder include the commencement of the urethra in its growth, and on more than one occasion the spongy part of the urethra invaded by superficial epithelioma.

**Tubercle.**—We have said that occasionally, in tuberculous disease of the kidney, the bladder is ultimately involved, and in one or two cases the commencement of the urethra has been also affected, In one of these the tuberculous ulceration of the membranous part had given rise to extravasation of urine. In another example the tuberculous ulcers extend through two thirds of the length of the canal towards the orifice.

# DISEASES OF THE MALE SEXUAL ORGANS

## TESTIS

**Malformation.**—The testis may be absent on one or both sides, or its duct may be undeveloped when the testis is present. In other cases the testes are imperfectly formed and small. It is, perhaps, more important to notice that this organ on one or both sides may fail to descend into the scrotum, so that it is found in some part of the line it naturally travels from the loin downwards, unless it deviates and goes a little astray. Such retention of the testicle is more frequent on the left side. Its cause is not always discoverable, but sometimes there are peritoneal adhesions which explain it; otherwise we may suppose that the *gubernaculum* is not developed properly, or that the testis is too large, or the inguinal canal too small. When retained in the canal it may give rise to anxiety, and it is said, especially by Goddard, that through exposure in this situation and consequent liability to injuries, such testes offer a greater proportion of morbid growths than those which attain to their proper situation in the scrotum. He asserts also that no sperm is secreted by these testes. This was so in a case we examined. Curling says that the subjects of this malposition are sterile.

**Atrophy.**—This may occur from actual disease of the organ, as will be presently mentioned, or simply from pressure on the cord affecting its nutrition; thus, after the long use of a truss, the testis sometimes wastes. The same may happen from a varicocele, or yet more frequently through the pressure of the fluid in a hydrocele.

**Inflammation or Orchitis.**—The most common form of this disease is connected with urethral inflammation, generally gonorrhœa, and seems, indeed, due immediately to a continuation of the inflammatory process from the urethra along the vas deferens to the testis; and thus it is that the epididymis is so frequently the first part, or that solely affected. The inflammation in these cases generally subsides, and therefore we rarely have an opportunity of examining the anatomical changes which the organ has undergone. In the few cases we have seen there was a mucoid fluid or pus collected in dilated ducts of the epididymis, while the tissue around was rigid and swollen. In the tubes of the testis there was excess of ill-formed epithelium. This condition ultimately leads to induration and contraction of the epididymis. At the same time usually the tunica vaginalis is inflamed and full of fluid, producing a gonorrhœal hydrocele. We have on four occasions met with suppuration of the epididymis in pyæmia; in these cases there was a considerable quantity of pus, running into the condition of abscess. The appearance was then very difficult to distinguish from tubercular inflammation, but the pus was more liquid, and the tissue around more brightly injected; chronic thickening, too, was absent; the disease also did not extend far along the *vas deferens.* When this part of the organ is alone affected the term "epididymitis" is used. Grave inflammation leading even to sloughing has occurred through injuries, as once when the whole length of the cord and testis was laid bare in a patient who was run over; but in inflammations superficially arising the testis generally escapes; being most liable to those chronic kinds of inflammation due to injury or extension of urethritis. Calculus or gonorrhœa may produce an inflammatory enlargement of the organ, an affection for which testes were frequently excised, and it is probable that some of our older specimens, which are deficient in history, present nothing more than this condition: one, for example, is said to have been growing slowly, in consequence of a blow five years before, but we have not examined it. Sometimes these chronic inflammations end in *suppuration.* The testis becomes adherent, and the scrotum is involved; the abscess then breaks, and the glandular structure becomes exposed, while spongy granulations protrude. This was formerly called *fungous testis,* and was generally removed by the surgeon. Now, however, these cases are viewed as simple inflammation, though there is some doubt whether some of them at least may not originate in a truly tuberculous epididymitis—at any rate, they are cured, except where, by a long inflammatory process, much lymph has become effused into the tissue, and the organ has thus been indurated or destroyed, when excision may be advisable. In such cases as these we have more than once had an opportunity of making a microscopical examination, and we have found remains of the seminiferous tubules enveloped in fibrous tissue, and their natural contents gone.

In most cases of orchitis, however, which come before us after death, the inflammation has long ago passed through all its active stages, and then what we find is a condition which resembles that called cirrhosis in the liver and lung. For the chief part of the section is seen to consist of new fibrous tissue, in which there are only remains of the natural structure with the septa thickened. The testicle is of the usual size, or generally rather smaller; it nearly always adheres to its *tunica vaginalis.* There is then often no history of the disease to be obtained, and we are left to infer from the associations what the probable cause of the orchitis may have been. These associations show that it is nearly always syphilitic.

Although orchitis is generally a local disease it may sometimes have a constitutional origin; the most common form being that associated with mumps. In exceptional cases of this complaint the testis becomes painful and inflamed for a few days. Paget has also described an orchitis coming on spontaneously at a later period of life, and which he believes to be gouty; sometimes it may be simply rheumatic. In various fevers orchitis has occasionally been observed as in typhoid, and Trousseau and others have seen it in smallpox.

**Syphilitic Orchitis—Syphilitic Sarcocele.**—The characteristic anatomical feature of syphilitic disease here, as elsewhere, is the "gumma," and this is, as usual, local—indeed, it has no peculiar features in this part, but resembles exactly the gumma found in the liver, &c., forming a yellowish, somewhat elastic knot, which used to be called a tubercle. It is always surrounded by an indurated fibrous zone, and is composed of interstitial inflammatory formation, with more or less evident remains of the natural tubular tissue all undergoing retrogressive changes. The disease is often one-sided, like other "tertiary" effects of syphilis. When the gumma is large it is sometimes difficult to distinguish it from a true tuberculous disease; but the following characters will generally guide us. Syphilitic gumma is fleshy or elastic compared with tubercle. It is surrounded by an area of callous fibrous induration. It occurs in the body of the testis, while tubercle affects the epididymis, or rather this is usually the case, for syphilis may affect the epididymis; again, it does not tend to soften and suppurate or implicate the scrotum, and break externally like tubercle, although it almost always causes adhesion between the testicle and its tunic—not invariably, however, for sometimes there is hydrocele. Tubercle, as we shall presently see, probably originates in the tubes, whilst the syphilitic process takes place between them. We have seen tubercular and syphilitic disease occur together in the testicle, each with its proper character; but the gumma is not always present in syphilitic orchitis. We have already said that most cases of fibrous wasting of the testis show by their associations a syphilitic origin. Such cases appear as a fibrous infiltration of the organ unequally diffused, affecting some bundles much and others little, so as to cause a mottled appearance of the organ on section, or else the whole organ may be fibrous. In nearly all cases of such fibrous change in the body of the testis we find other proofs of the presence of tertiary syphilis, so that in every post-mortem on a male subject the testis will offer convenient and ready indication of syphilitic taint. Though we must remember that, whatever reliance may be placed in the inference from the presence of this fibrous change, we must not set down a case as free from syphilis because the testis has escaped. It is sometimes met with in infants as a congenital disease, and even in the new born.

**Tubercle.**—The testis proper sometimes contains a few tubercles in cases of general tuberculosis, though this has been denied. But when the organ is primarily affected the disease shows itself first almost invariably in the epididymis, which is found swollen greatly and converted to a caseous mass, more or less softened and changed to curdy pus, which is contained in spaces loculated with the relics of the walls of the tubes. We might remark that many specimens in the Museum showing hard yellow masses in the testis, and which were formerly called scrofulous, are no doubt syphilitic. The exact seat of origin of tubercular disease has been much contested, as a part of the general question whether it belongs to the ducts or the interstitial textures of glandular organs, some holding that the process primarily concerns the mucous surface of the tubes, while others think its proper seat is the interstitial tissue. The authority of Virchow is given generally to the latter view, and accordingly he holds that it is in the connective substances in and around the tube walls that the tuberculous matter appears, while he thinks there may be catarrhal products within the tubes secondarily. Others maintain that in the very early process tubercles may be seen in the walls of the tubes, and that they have originated within by a proliferation of the epithelium. They are composed of a mass of cells, in which are seen giant-cells. There are still other observers who believe the origin of the tuberculous disease is to be found in the lymphatics.

In one case there was a cheesy mass in the epididymis, whilst nodules were scattered through the body of the testes, and granulations in the walls of the tubes occupied their interior. Bacilli were present. Whatever view may be held, the appearance in an early stage shows the tubes of the epididymis filled with caseous pus, but the walls are also caseous. Around the more advanced disease we often find more or fewer miliary tubercles in the *rete* or in the testis proper, near the epididymis. On examining these we find evidence that they do arise in the interstitial substance, but whether this was the case in the earlier and now more advanced disease in the epididymis we never could be sure. Such tuberculous abscesses of the epididymis may contract adhesion to the scrotum and open outwards, discharging their contents, while the organ in favorable cases shrivels and may cicatrize, or a fistulous channel may remain, from which exuberant granulations may sprout and deserve the old name of "fungous testis, or "strumous testis."

The disease always extends along the *vas deferens*, converting this into a thick cord with greyish pellucid walls and a caseous centre. Thus the prostate may be reached, and the tuberculous process may there acquire fresh force, so that caseous prostatic abscesses form and even may lead to perforation outwards, or fistulous communication with the bladder. The *vesicula seminalis* of the same side shares in the disease, and thus there is tuberculous disease of the lining of the whole genital system on one side ; sometimes the bladder, ureter, and kidney are also involved. But the testis proper is late in being implicoted, and then usually only shows a few miliary granulations, irradiating from the primary seat of disease in the epididymis. Sometimes the miliary tuberculosis arising from tuberculous epididymis becomes general and fatal; of this we have seen several instances. It seems true that tuberculous disease may proceed downwards from the kidney to the bladder, urethra, and neighbouring parts, as well as sometimes pursue a course upwards in the opposite direction.

**Morbid Growths.**—*Fibroma.*—We have had occasion to observe before that chronic inflammatory products and tumours are not always so easily separable as might be thought. Thus we have just now spoken of specimens of testis in which the enlargement was due to a fibrous element diffused through the tissue. There is some difficulty in knowing whether it be better to call them enlargements from chronic inflammation or fibrous tumours. Generally, however, no such difficulty exists, for if an inflammation can be distinguished from a new growth by the one being an affection implicating intimately the whole tissue, and the other a growth increasing from a centre of its own, then, in many of the cases, the term chronic inflammation or fibrous tumour is easily applied in the respective examples, especially, too, if the tumour occupies a small part of the organ, while the true glandular structure is pushed aside or stretched over it.

We sometimes meet with small polypoid growths of fibrous tissue on the capsule of the testis, especially in cases of hydrocele.

*Sarcoma* is not frequent in the testis in a pure form; if so, it begins in the body of the organ, and gradually grows either as a circumscribed tumour or in a more diffused manner; as a rule we are soon struck with the curious proneness of growths in this organ to show a polymorphous structure. The commingling of several varieties of structural elements in tumours of the testicle makes it difficult to refer the tumours that occur in it to our ordinary classes of morbid growth. Thus we find cartilage mixed with medullary sarcoma in a tumour that has numerous cysts in it, and although this is called a compound enchondroma, yet we must not, in so naming it, overlook this curious association of textures not elsewhere found together. Again, we may find mucous tissue, cartilage, glandular acini, and large cysts together in a recurrent tumour of the testis, and be much at loss to know what class to refer such a growth to. We do meet with testis-tumours of a sarcomatous nature, the texture being of the spindle-celled variety; but most commonly there are cysts present and the cells have communicating processes which make a meshwork in which a mucous fluid is contained, so composing cystic myxosarcoma.

Both the *round-celled* and *spindle-celled* sarcomas appear to originate in the intertubular connective tissue. In some cases the cells so much resemble the glandular tissue that the name *adeno-sarcoma* has been given to them; several such have been exhibited at the Pathological Society.

*Cystic myxosarcoma or "cystic testicle"* is a name given to tumours of the testis that are chiefly composed of cysts. The solid part of the structure varies much; they grow to a large size. In specimens which are characteristic examples we found the stroma to resemble the tissue of which nasal polypi are composed; that is, a lax "fibro-cellular" (myxosarcomatous) web, made up of spindle-formed and multipolar cells connected by a filamentous meshwork in which is a submucous fluid. There were, however, in some parts numbers of free lymphoid or epithelioid cells, while in other parts the texture was condensed to an almost purely fibrous structure. The cysts of various sizes contained either serum or mucous or colloid fluid. The tumours of the testis, composed of simple cysts with fibro-cellular walls, generally are "benignant," and do not recur on removal. But all cystic tumours of the testis are not so simple; some, curiously, contain muscular fibre, striated or non-striated, or cartilage, or else they may contain cancerous tissue, or all these together, and then prove malignant, recurring after removal. We have thus both innocent and malignant cystic tumours. As to the origin of the cysts themselves, they are mostly new formations, but sometimes are dilated tubules. Curling believed that the cysts were produced by dilatation of the tubules in the rete testis; and Eve agrees with this, showing that dilated portions of tubes are cut off by folds and then expand. The cysts are lined with epithelium occasionally columnar. Eve thinks with Bland Sutton that many of these tumours have their homologue in the ovary and originate in the hilum, where are found the remains of the Wolffian body. From these fœtal rudiments the cystic growth springs, and this accounts for the fact of the testis being sometimes found intact spread over the tumour. Sir J. Paget, in a remarkable case of enchondroma, in which the cartilage grew in cystic spaces, found that these spaces were dilated lymphatics.

*Myxoma* occasionally is found as a large, soft, mucous tumour of the testicle, described by Lebert as fibro-colloid.

*Carcinoma.*—Cancer varies in different parts of the body according to the texture in which it occurs; and thus, although there is such a form as medullary with only a very tender matrix, and scirrhous with a firm and abundant one, yet the differences of these are due in great measure to the character of the organ in which the disease occurs; thus, in the dense fibrous structure of the breast cancer is scirrhous, while in the testis it is *medullary*. We see many examples of this disease on our shelves; the organ is much enlarged, and, when cut, is found to consist of a soft matter, which is well called encephaloid, for it very closely resembles the structure of the brain, giving out a milky juice, and the microscope showing a predominance of cells contained in a very delicate stroma. Remains of the testis will generally be found on the surface, having been pushed away by the increasing growth; and we often find yellow deposits of degenerating fibrin as well as these yellowish-white masses of the decaying cancer itself. Cysts occur in carcinomatous testes, and may be so much developed as to reduce the carcinoma to the relation of an interstitial material.

These are sometimes full of blood. Sometimes cartilage is present in these tumours and even at a very early period of childhood. They expand the tunica albuginea, and often set up secondary tumours of the iliac and lumbar glands.

It has not yet been positively settled in what structures cancer of the testis begins, but many good observers maintain that it commences in the epithelium of the tubes which become distended with cell-growth, and that they subsequently rupture, allowing the new growth to proceed amongst them and ultimately destroy them.

*Scirrhus,* or hard carcinoma, is rare in the testis, but a few well-marked cases have occurred, as one in our Museum removed by Mr. Bryant. The structure is characteristic.

*Enchondroma.*—This is a very remarkable, though not uncommon, disease of the testis ; and we possess a very good specimen of it. The organ is enlarged, and in it are translucent nodules which under the microscope have the appearance of well-formed cartilage. But we observe that this cartilage does not form a continuous mass ; it appears as a number of distinct pieces, separated from each other by an intervening substance of fibro-cellular character, and thus it is very different from the pure enchondroma so common in the bones. The origin of cartilage in the testis is very curious ; it is a good example of *heteroplastic* growth as defined by Virchow ; that is, of the rise of a tissue widely different in character from the matrix in which it takes origin. Also the enchondromatous testis well illustrates Virchow's teaching that such heteroplastic origin brings with it malignancy, for while cartilaginous tumours on the bones—natural seats of cartilage—are generally innocent, a great suspicion surrounds the character of any cartilaginous tumour of the testicle. In a case which occurred to Sir J. Paget, which appeared to be pure enchondroma of the testis, the cartilage returned as a secondary growth in the lymphatic glands and in the lungs. We possess a small portion of the lung which is consolidated by it. The cartilage is, however, very rarely pure, but rather, as in the case mentioned, it is combined with other morbid material, either myxomatous, sarcomatous, or even not unfrequently carcinomatous. In the return of such tumours after removal cartilage generally grows also in the new mass, which appears usually in the iliac glands or in the viscera, although we have known such a compound cartilaginous growth return in the scrotum. One interesting particular in these growths is the development of the cartilage within hollow spaces, cystic or tubular, in which nodules and rods of cartilage lie apparently free. Paget found these spaces to be dilated lymphatics, and traced the cartilage up the lymphatics of the spermatic cord. Virchow saw the cartilaginous growths both in the blood-vessels and lymphatics. Quekett described them as in the ducts. We have found them in lymphatic spaces, while some dilated ducts contained curiously formed compact masses of epithelium very like fœtal cartilage. The growths in the lymphatics at first appear as if they were free, but closer examination shows them to be connected with the vessel's wall, from which they grow as polypoid masses, projecting into the dilated channel.

Wherever cartilage is formed we are likely to have osseous tissue, and thus we might not unreasonably expect to meet sometimes with bone in these diseased testes. In the lower animals, we believe, complete ossification of the testis sometimes takes place.

Butlin, in his lectures at the College of Surgeons, stated that although children often had malignant disease of the testis, it was more usually sarcoma than carcinoma, and was both of the round-celled and spindle-celled variety. He believed the latter was more common in the young, whilst the former occurred at all ages. He was also disposed to think that enchondroma was not malignant, and that Paget's well-known case must have had some other elements interspersed with it. Tumours of the testes occur at all ages from birth to senile decay.

*Dermoid cysts* have been found in the testes containing hair and sebaceous matter, and more rarely, teeth; indeed, all the substances met with in similar cysts in the ovary.

*Hydatids* have now and then been met with.

## TUNICA VAGINALIS

**Inflammation.**—Inflammation of the tunica vaginalis with production of lymph is generally artificial, caused by injection in order to obliterate the cavity after tapping for hydrocele. If by chance we get an opportunity of seeing the membrane a few days after the operation we find it showing, very well, the usual characters of acute serous inflammation; the cavity in a case we inspected was filled with soft yellow lymph, and the surface of the testis covered with a firmer layer. Such inflammation is very rare as a primary disease.

**Hydrocele**, or collection of serum in the tunica vaginalis, must be ascribed to a chronic inflammation of that membrane. In cases of great anasarca, even when the scrotum is largely implicated, the sac of the tunic is almost free from fluid, so that we cannot regard hydrocele as dropsical. Again, the disease resembles in character a chronic pleuritic effusion, and when it is tapped we find that the fluid from it coagulates.

If the portion of peritoneum which carries the testes into the scrotum is not closed, the sac communicates with the peritoneal cavity, and then if distended with fluid is called *congenital hydrocele.* Again, if the vaginal process of peritoneum is obliterated only at its upper and lower part, a space will be left along the cord which, if filled with fluid, may distend the scrotum and become an *encysted hydrocele.* This term has also been used for cysts which primarily form on the testis or its appendages, but for the latter, it is better, in order to save confusion, to adopt the name spermatocele, although the latter was used by old writers as synonymous with varicocele.

The tunica vaginalis generally may be found thin and pellucid; but if the case is of long standing, and the part has been subjected to much irritation or injury, as is often the case, we find in it the results of chronic inflammation, so that the serous membrane has become very much, and sometimes enormously, *thickened*; in some of these cases it is a question whether the inflammation may not have been due to curative measures formerly adopted, for we do not think the serous cavity is always obliterated by injection. The sac may be greatly thickened, while the testis is reduced to a small size by pressure of the fluid. The thickening of the sac is often not uniform. Sometimes there are merely indurated spots on, or hard *nodular bodies* projecting from, its inner surface or that of the testis; these occasionally become *cretaceous*, and, indeed, the whole membrane may become bony or calcified. The depending fibrous bodies which form on the testis may sometimes drop off, as occurs in the joints, and may be found loose in the tunica vaginalis. In one or two such which we have examined the texture was not true bone. Mr. Shattock reported a case where the fluid drawn off was milky or chylous.

**Hæmatocele.**—This name is given when the tunica vaginalis becomes filled with blood, a state generally arising from injury. The blood usually becomes absorbed, and no injurious result follows; sometimes the fluid parts are taken up, and the fibrin forms distinct layers, resembling the interior of an aneurysm; in the more unfavorable cases the blood softens down, and a sanguineous cyst is produced; in such a case, if the cavity be tapped a fluid is drawn off corresponding to changed blood in other parts—viz. chocolate-coloured fluid, containing numerous plates of cholesterine and hæmatoidin crystals, as we have before described. Very often a hæmatocele is produced by injury to a hydrocele, either in tapping, or by accidental blows, or inflammatory rupture of vessels. The false membrane sometimes found suggests an inflammation in the first place followed by effusion; it being too generally assumed that the membranes result from blood.

A difficulty in diagnosis sometimes occurs in distinguishing hæmatoceles from collections of blood in the testis itself; for sometimes both sarcomatous and carcinomatous growths contain sanguineous cysts.

**Spermatocele or Spermatic Hydrocele.**—Sometimes cysts form at the upper part of the testicle about the epididymis. One or more of them may grow into a large size and thus closely resemble common hydrocele of the tunica vaginalis, hence it is sometimes called *encysted hydrocele*. But the fluid drawn from them is apt to be milky from admixture of semen, whence the name spermatocele is a preferable one for such cysts. The exact origin of the cysts is a subject of great interest; much light has been thrown upon it, yet some discrepancy of views still remains; but they are now almost universally regarded as diseased enlargements of the remains of the Wolffian body. A part of this as is well known becomes converted into the epididymis, while its duct forms the *vas deferens;* but some parts of the body remain unused. They then correspond to the cysts of the parovarium of the female. It is thought by Virchow that such remainders, being still in connection with the epididymis which arose from parts contiguous with them, may enlarge and form cysts into which the spermatic fluid regurgitates, perhaps through obstruction of the *vas deferens.* Paget suggested that the cysts were new growths and produced semen in virtue of their nearness to the testes. Curling argued against this view that semen is not found in the cysts when small, as would be expected, but when very large; and we know that large cysts in glands generally cease secreting even when they arise by dilatation of the proper secreting sacs of the gland. The fact that semen only appears in the larger cysts is equally against Virchow's views. We too have found that the semen is not present in the small cysts, and hence believe, with Curling, that it probably enters the large cysts during their growth through opening of the ducts by their pressure, just as bile enters an hydatid cyst.

The cysts may be more or less numerous; sometimes a single cyst is embedded in the epididymis; and this is rarely multilocular; they are lined by a beautiful squamous epithelium very separable from their wall, and they arise in the subserous tissue of the epididymis, or of the neighbouring part of the tunica vaginalis, quite unconnected with the epididymis. The mass of cysts may make a considerable tumour above the testicle.

## VAS DEFERENS AND SPERMATIC CORD

**Hydrocele of the Cord,** a collection of fluid in an abnormally persistent portion of the *processus vaginalis,* sometimes occurs as a distinct form of disease, as may be seen in a specimen on our shelves; the inflammatory changes which we have described may then occur in the serous membrane, producing thickening or even earthy degeneration, as in this case.

*Encysted hydrocele of the cord* has already been spoken of; it has also been called hydrocele of the processus vaginalis.

**Varicocele** is a term given to a varicose condition of the spermatic veins, which form a large tortuous bunch of twisted cords with walls much thickened at the lower part of the testis; sometimes we find thrombus in a varicocele. Sometimes the vessels become ossified, and we have seen them forming a hard mass, associated with a similar cretification of the iliac and pelvic veins. The *vas deferens* may also sometimes be found ossified.

Opinions vary as to the causes of varicocele. Like varicose veins in the leg there is no proof of any pressure on the veins above. It is usually seen in young people, and begins often about the time of puberty, as if an increased vascular impulse towards the genital organs operated in the production of a venous hypertrophy. It mostly occurs on the left side, which is generally considered to be due to the mode in which the spermatic vein enters the renal. There is also a question with respect to any interference with the nutrition of the testis, but most authors are inclined to agree with Sir A. Cooper that the testis is soft and relaxed, and somewhat smaller than the other. In some cases where the varicocele has commenced in youth the testis has remained small, but it has also been observed that when the affection has come on at a later period of life, occasionally the testis, from being before of normal size, has become wasted.

**Tubercle** may extend along the cord, when the same form of disease exists in the testis.

**Cancer,** in the same manner may involve the cord, as well as *enchondroma* and *melanoma*, but these are rarely primary diseases. Several cases are recorded of *sarcomatous* tumours growing on the spermatic cords of children; these are often mixed with fat, or may be wholly fat, and therefore styled *lipomatous*. *Myomata* have been described as having their origin in the cremaster muscle.

## VESICULÆ SEMINALES

**Atrophy** of these organs occurs often in old age, and the enlargement of the cells, which is often seen in the aged, is probably a species of degeneration.

**Inflammation and Suppuration** is not unfrequently met with in cases of pericystitis and abscess of the prostate, where the base of the bladder is involved.

**Tubercle.**—In cases of tuberculosis of the testis and spermatic cord, the vesiculæ seminales may be affected; often only one is involved, when the disease of the testis is unilateral.

**Carcinoma,** when affecting the neighbouring organs, may involve also the seminal vesicles. In one example reticular carcinoma arose from the vesicles themselves, extending into the tissues around.

**Concretions** may also be met with in them.

## PROSTATE

**Hypertrophy.**—This is the most common and important disease of this body, especially when it leads to outgrowth of a portion of the organ into the bladder, constituting what is called the middle lobe. The prostate may be uniformly enlarged, but such a uniform hypertrophy of the whole organ is rare; for we shall nearly always find on section of an enlarged prostate that the increase is due to a formation of one or more circumscribed tumours in its substance, and this even when on the exterior the organ appears to be equally augmented. They have sometimes been turned out in the operation of lithotomy. The tumours or myomata are of the same constitution as the prostate itself. This is composed of glandular tissue, sparingly present in a fibro-muscular mass, which acts as sphincter to the bladder. According to Virchow we may find the glandular, the fibrous, or the muscular matters to predominate in the new growth, while the general character is maintained. This is true, and is a matter of pathological interest; sometimes, indeed, we find a small quantity of glandular product in sections of such tumours. But yet the main run of cases show among themselves very much the same kind of material, and this is very like that of fibroid tumour of the uterus. When we recollect how close are the homological relations of the uterus and prostate this must arrest our interest, although it is not quite true that the prostate is in reality the masculine equivalent of a uterus. For homology to the uterus is strictly limited to the *vesicula prostatica*, yet, certainly, the behaviour of the uterus and prostate as to these myomatous growths is curiously alike. In each organ they come as distinct tumours in declining life, and they have in the two almost identical characters. The favourite seat of them is in the thicker part of the base of the prostate at the neck of the bladder behind the urethra, where, rising up, they form valve-like masses that too effectually close the bladder, so that at last there is no way out of the cavity except by tunnelling the prostatic lobe with the catheter; such a mass is often called a "large middle lobe," but it is really a new growth, and may be found separate from the main mass of the organ: these enlargements are rare in the upper part of the prostate. The effect on the prostatic part of the urethra is to flatten its channel, while at the same time the extent of the urethral surface is greatly increased, so that, for instance, we may find the canal three inches around, but flattened closely. The channel may be bent out of its ordinary course, but its great width prevents any real stricture as a consequence of this deviation.

Although such enlargements probably occur more frequently in advanced years than in the middle period of life, yet they by no means are necessarily connected with age; for many years past we have been in the habit of examining the prostate in old persons, and have found that, speaking generally, its enlargement only occurs in those cases where it produces symptoms and has been a source of complaint during life. We can, therefore, quite confirm the statement of Sir Henry Thompson, that enlargement of this organ is not a necessary attendant on old age; indeed, we have found it sometimes rather atrophied. We do not mean that this enlargement is not more frequent in advancing age, but it is not one of its necessary or ordinary concomitants, as grey hair or calcified blood-vessels, though this was formerly thought to be the case.

**Fibroid Degeneration.**—So far from enlargement necessarily occurring in old age, we have sometimes found the organ atrophied. Also in younger people we have met once or twice with a condition which might be called fibroid degeneration; and in one case this was associated with a similar affection of the testis, which, we have no doubt, was due to syphilitic cachexia. The organ was converted into an amorphous hard substance, interspersed with opaque white parts indicating a fatty and granular degeneration.

**Inflammation and Abscess.**—This is a not uncommon affection, coming before the surgeon either as an acute inflammation caused by gonorrhœa or injuries, or else as an old disease in connection with stricture, when it is frequently found in the post-mortem room. In long-standing cases of stricture, where perhaps urinary fistulæ have long existed, and especially where false passages are present, and the patient has died from some acute mischief about the part, either with or without external extravasation of urine, the prostate and coats of the bladder are often found involved in a suppurative process. There may be fistulous connection of the urethra with the bladder or rectum, or an extension of the fistulæ under the peritoneum, or into the perinæum. It is difficult to say, when such mischief has occurred, what has immediately set it up—whether it be a mere continuation of the urethritis backwards, or the effect of injury done to the prostate by catheters, but we find openings on either side passing into this organ; and when the lobes are divided they are found full of pus, or the whole structure, perhaps, is in a sloughy condition.

We sometimes find the prostatic urethra widened much in cases of stricture by the pressure of the urine. In one of our cases it is sacculated on each side, apparently from the same cause.

We possess a specimen of prostate, which was removed from a man who had been operated on for lithotomy fourteen years before, and in which not the slightest trace of the incision can be seen. But cases are recorded where not only scars were observable but atrophy of the gland.

**Tubercle.**—This is not usually a primary disease, but merely a part of a general tuberculosis of the urinary or genital organs; but the prostate may be extensively infiltrated, and the tuberculous mass may soften down into large cavities, which become a source of much suffering to the patient.

**Cancer** of the prostate is very rare, but it does sometimes occur as a primary disease. Several cases have come under our observation, once of soft, twice of firm reticular carcinoma. The disease is not generally limited to the prostate, but has a remarkable tendency to extend to the tissue around. In one case the growth justified the name "lympho-sarcoma."

We sometimes meet with simple *cysts* in the prostate; we have a specimen of one opening into the urethra. They usually contain concretions. In a case which occurred lately of vesical calculus, there was a growth in the prostate composed of several cysts. It resembled in character the fibro-cystic tumour of the uterus.

**Concretions or Calculi.**—These may be found, on making a section of the prostate, as small yellow translucent bodies like amber, or sometimes as blackish grains; when large they are facetted by mutual apposition and friction like gall-stones. This would distinguish such concretions from any other kind of calculi from the urinary passages. Sometimes, however, large calculi form in the organ, where also they are endeavouring to escape into the urethra.

## PENIS AND SCROTUM

**Malformation.**—Some important malformations of these organs are connected with extroversion of the bladder. In *Hypospadias* the urethra remains open on its lower surface for a greater or less extent, along with a proportionate degree of smallness and general incompleteness of the parts. In its extreme degree the urethra is open all along the scrotum, which is thus divided into two halves, while the testes, of small size, are retained more or less above their proper position.

**Inflammation.**—The surface of the glans is liable to simple and gonorrhœal inflammation, or *Balanitis*, producing pus between it and the prepuce; if the latter be closed by Phymosis the matter may collect and cause ulceration, and perhaps perforation, or even gangrene of the prepuce.

Sometimes the body of the penis inflames, and the *corpora cavernosa* are found to contain lymph or pus. This is not frequent; it may result occasionally from injuries or severe gonorrhœa, or it may arise, as in other exposed parts, through putrid fevers. The effect may be general pyæmia; or destruction of the part; or fibrous wasting. Occasionally calcareous matter has been known to be deposited in old fibrous patches. Sometimes in old dropsy cases we find pus in the scrotum.

The terrible inflammation of the external genitalia produced by extravasation of urine, is too familiar to us in the wards, and we see also the means by which the fluid is prevented from descending upon the thighs, through the union of the superficial fascia with Poupart's ligament, and the pubic arch.

*Erysipelas.*—We have seen several cases in which all the external characters of extravasation of urine have been present in the form of *gangrenous* erysipelatous inflammation in this region, but without any disease or injury of the urethra at all, so that the disease proved to be a *simple erysipelas* of those parts.

*Syphilitic inflammation.*—It is not often that we meet with open chancres post mortem. The peculiarities of syphilitic chancre, their greyish indolent surface, and congested callous margin all belong to the living condition. The induration alone remains after death. This induration, we know, may persist some time after the healing of the sore. The material causing the induration is composed of roundish cells similar to those that make up the substance of granulations in healing wounds, so that Virchow classes it with "Granuloma." We should accustom ourselves to be quick in recognising scars of bygone syphilitic sores. Should a scar of chancre be present we should always look for the scar of a bubo. The presence of the latter takes away nearly all the weight of the former as evidence of syphilis, since, as we know, chancres that cause suppurating buboes very rarely cause syphilis, though they are sometimes found associated.

*Eczema, herpes, boils,* &c., occur on the penis, as elsewhere on the skin.

*Condylomata* are frequently found on the external genitalia, both the *acuminated* form, due to simple or gonorrhœal irritation, and the *broad* form, which is truly syphilitic. Condylomata of the first kind arise in, and take their character from, the papillary layer of the corium, while those of the second consist of the deeper skin infiltrated and raised into elevations by the same granulation-like material which is found in the base of the hard chancre, and which Virchow calls granuloma.

**Epithelioma.**—This on the penis generally assumes a warty character, and thus very large "cauliflower excrescences" are produced on the organ. Such a term is peculiarly applicable to many specimens in our Museum; while in others we see the disease at its earlier stage, where merely the adjacent parts of the glans and prepuce are affected. The structure shows very plainly all the characters of epithelial cancer. We do not know that surgeons have yet agreed as to its greater frequency in those who have been the subjects of congenital phymosis, but we can well believe how any unnatural condition should excite the disease, and especially where any irritating secretions are retained. Like epithelioma elsewhere, this is a local affection, and when removed early and with sufficient margin, does not return. If it be allowed to continue the disease involves the glands. On examining the body we may find that the more superficial have contaminated some of the deeper glands, and a few around the iliac vessels may be implicated, but, apart from this, no disease will be found in the body.

*Carcinoma.*—We not unfrequently meet with well-marked carcinoma of the penis; we have examined several specimens. The structure of the growth has generally been markedly of an adenoid type, the acini being often very well formed, but the cells were large, multiform, and with large bright polynucleolated nuclei. Mr. Golding-Bird had a case of well-marked spindle-celled sarcoma.

*Cancer, or epithelioma of the scrotum,* formerly called *chimney-sweepers'* cancer, is the same kind of disease as that just mentioned, but affecting the skin of the scrotum; it is caused especially by the irritation of soot, and is now, we believe, much less frequent than formerly; it begins as a wart, then spreads, and subsequently ulcerates until a round raised raw surface is seen on the scrotum, having an ichorous discharge; when excised early a perfect cure results; when not, the inguinal glands become fatally affected in the same manner as we have just mentioned. But very different is the result of carcinoma of the testis, which, being of the purely medullary kind, very soon involves the lumbar glands, and the patient dies with large tumours in his abdominal and other organs.

**Chronic Hypertrophy of the Skin and Cellular Tissue, or Elephantiasis Scroti.**—This is the same kind of disorder which occurs in the leg under the designation Elephas, or Elephantiasis Arabum. It consists of a simple hypertrophy of the tegumentary tissues. It may affect chiefly the penis or the scrotum, but generally reaches its largest development in the latter, dragging the integument and prepuce down, so as to produce distressing impediment to micturition. We may see by our Chinese pictures to what an immense size these tumours grow in the East; this is partly due to the want of surgical skill to remove them when small. One of the most remarkable is that in the Museum, which was removed by Mr. Key from Hoo Loo, a Chinaman; it weighs several pounds. A disease alike in kind, but very different in degree, is frequent enough in this country.

In some cases however, when the scrotal tissues are swollen, as in elephantiasis, there are found sarcomatous elements mixed with the hypertrophied connective substance.

We have already alluded to *lymph scrotum,* or those cases where there has been a milky or lymph discharge, containing within it the *Filaria sanguinis.*

The teguments of the scrotum have been found distended by decaying blood-clot, so as to form a tumour.

We have already spoken of a scrotum containing *chalky deposit* in a gouty subject.

*Mamma.*—The male mammary gland is subject to the same diseases as the female, though to a less extent. Thus our Museum contains several specimens of carcinomatous tumours, which resemble the female type in being scirrhous; sarcomas, however, are sometimes met with.

# DISEASES OF THE FEMALE SEXUAL ORGANS

## VULVA

The extreme degrees of malformation in which the external genitalia are quite absent or very imperfect are not of practical interest, as they occur in unviable fœtuses only.

**Malformation.**—We have seen that in hermaphrodites the genitalia may resemble female organs as to external conformation, while the individual is essentially male in the possession of testes. So likewise we find that when the individual is essentially female in the possession of ovaries the external organs of generation may approach in superficial characters the appearance of the male. Thus the clitoris may be long, and pierced by the urethra, while the vagina becomes small and the uterus and ovaries imperfect, the latter, perhaps, descending into the labia.

The labia or nymphæ may grow to too great a size, or may be united together.

**Inflammation.**—Skin diseases become localised about the vulva; thus eczema, herpes, erythema, erysipelas, &c., are obstinate there; also lupus may appear in its several forms. *Noma* or gangrenous phlegmon, which we have described as it appears in the cheek, is also found here, more frequently in children.

**Injuries** may arise during a severe labour, owing to pressure of the child's head, by which a large vessel, generally a vein, is lacerated; blood is effused, and a great tumour is formed; if this be opened, altered blood is discharged. We may see similar swellings in the surgical wards, arising from direct blows, and they occasionally appear to have been caused by strains. Such swellings may suppurate and even cause death by extension of the inflammation to the interior of the pelvis. Varices of the labium are not infrequent in pregnant women.

**Œdema** may be a result of pressure during pregnancy, or may arise in general dropsy, being in some cases excessive when the dropsy is slight elsewhere. When œdema becomes extreme the part may inflame and suppurate, and there is danger of gangrene. A *gangrenous* condition may also be set up during putrid fevers.

**Morbid Growths.**—*Condylomata*, both acuminated and broad, such as we have described in the male genitalia, are constantly met with also about the vulva. The former, due to simple irritations or gonorrhœa, may be of a simple kind, like those which occur on the hands, though they often form large masses; the latter are syphilitic; they too may grow to a large size. We also meet with conditions corresponding to the elephantiasis scroti of the male, consisting indeed of similar hypertrophy of the skin and cellular tissue. This form of disease may here put on the shape of a polypoid outgrowth, or may have a broader base and be continuous with the labium. On our shelves may be seen a large tumour from the labia, and another from the clitoris.

*Epithelioma*, or epithelial cancer, sometimes attacks these parts, but it is not a very common affection. It shows the usual characters of this disease. We have seen it on the clitoris of a warty character and exactly resembling the same disease on the penis.

*Lupus* occasionally attacks the genital organs. It may appear externally in the form of nodules or lupoid tubercles or in the vagina as an ulcer with thick raised edges. Some cases of very chronic disease of the cervix have been thought to be lupoid in character, and instances have been reported where lupus was supposed to have affected the body of the uterus itself.

*Fibrous tumours* occur in the vulva as in other parts, and are frequently removed by the surgeon. They are composed of dense cellular tissue like the labium itself, and may be present in any part of the vulva, sometimes growing to a great size. They differ from elephantiasis in being circumscribed, and not indefinitely continuous with the general subcutaneous textures.

*Lymphomatous* tumours also occur here occasionally, sometimes combined with *fibroma*.

*Encysted tumours* may also sometimes be met with here; we have seen several such, and they contained a limpid, watery, or a mucous fluid. Dermoid cysts sometimes appear also. We have already spoken of blood-cysts and abscesses.

*Hernia.*—We may just allude to tumour in the labium produced by inguinal hernia.

## VAGINA

**Malformation.**—In some cases of imperforate anus the rectum opens into the vagina. Sometimes the vagina is double, as in a case where the uterus is double also; a double uterus generally indeed accompanies this state, but sometimes the vagina is nearly divided when the uterus is single. Again, besides sharing in the faults in the direction of hermaphroditism, in which the vulva is chiefly concerned, the vagina may be absent, either when the uterus is also absent, or in the presence of the uterus. The whole canal may be closed up, or only its upper or lower part, or both. Sometimes such congenital occlusion of the vagina seems to be the result of inflammation during fœtal life, for it is found when the development of other parts shows no failure. It then resembles the results of severe ulcerative inflammation in early life, which likewise may produce almost or entirely complete occlusion of the passage.

Sometimes the vagina is closed by an *imperforate hymen*, when an operation may have to be performed for retention of menses, and a large quantity of dark pitchy fluid, like treacle, consisting of altered blood, mixed with mucus, may be drawn off.

**Variations in Size.**—*Prolapsus, &c.*—Dilatation occurs above the obstruction in the cases of retained menses above described, or through prolapsus uteri, &c. When there is dilatation or loss of tone, as after parturition, the vagina itself may prolapse either around all its circumference, like prolapsus ani, or more frequently by its anterior or posterior wall. When the anterior wall comes down the bladder may be brought with it, forming a *cystocele;* or the rectum when the posterior wall descends. Sometimes the small intestines in Douglas's pouch force in the back of the tube, and protrude into the vagina or at the vulva, forming an *intestinal vaginal hernia.* The uterus descends somewhat in any of these protrusions of the vagina.

*Contraction* of the vagina is caused by pressure outside it or by cysts, &c., in its wall, or through cicatrices of ulceration within. The tube may be lengthened and narrowed by the dragging up of the uterus adherent to a tumour growing out of the pelvis.

**Inflammation.**—Vaginitis shows itself generally as a catarrh, which is usually gonorrhœal. But there are other idiopathic forms of *vaginitis* which correspond to the varieties of stomatitis; thus, in children out-patients, an inflammation accompanied by a discharge is very frequently seen, which is often very difficult to distinguish from gonorrhœa. Also *diphtheritic* or pellicular inflammation is found in the vagina, generally in severe febrile exhaustion after parturition, or in putrid fevers, or in diphtheria itself; *ulceration* or *gangrene* may occur beneath the membranous layer; syphilitic ulceration we need scarcely mention. We meet too here, as in the mouth, with a rapid *gangrene* or *sloughing.* This occurs in the external genitalia of children, and the same name, *noma,* has been given to it; it has sometimes appeared as an endemic in particular localities, when its nature cannot be mistaken; but, meeting with an isolated case, we might for a moment believe it to have been the result of violence. We must be quite prepared for any such suggestion, and remember that, amongst the poor, where there is often a common sleeping-room for many individuals, a charge of violence is very apt to be made; but in these cases of idiopathic inflammation it is rather the vulva and external parts than the vagina which is affected. A case which occurred here a few years ago might have given considerable difficulty in diagnosis had there been any suspicion of violence; for not only were the labia and adjacent parts mortified, but owing to the purpuric condition of the body, which had led to the disease, large livid marks, exactly resembling bruises, existed on the arms and legs. Like cancrum oris, it is generally a consequence of measles, or one of the exanthemata. Gangrene may of course arise from direct injury or during labour.

*Aphthous inflammation* sometimes occurs in the vagina and vulva, as in the mouth, and it forms a very troublesome affection in women. The mucous membrane is inflamed, red, and covered with small ulcers.

We need scarcely allude to *leucorrhœa*, except as a reminder of the advantage sometimes gained by examining these discharges by the microscope; sometimes the knowledge so obtained may be of great service. The white discharge from the vagina contains merely mucous corpuscles and more or less perfectly formed pavement epithelium, while that from the os is more gelatinous, and if it has escaped from the cervix may contain cylinder epithelium.

**Morbid Growths.**—*Warts or warty tumours* are sometimes met with in the vagina, as will be observed in our wax models.

*Myoma.*—Tumours just like in structure to the ordinary uterine fibroid occasionally but rarely form in the vagina, especially the posterior wall. Sometimes when in the upper part they have arisen originally in continuity with the uterine fibres, but detached themselves afterwards during growth, in the same way as many subperitoneal uterine fibroids do. Such tumours may project in a polypoid manner.

*Mucous polypi* grow sometimes from the walls of the vagina. In a case we not long ago examined, where a uterine polypus existed, another was found growing from the vagina; and in another case, a polypus which had been removed by ligature sprang altogether from the vagina; it was as soft and vascular as the ordinary uterine polypi. These are not very common.

*Cancer* is unusual as a primary disease; but in cases of cancer of the uterus the upper part of the canal is generally involved. In the rarer form of disease, *epithelioma* of the external parts, the lower end may be involved.

*Cysts*, of which we have specimens in the Museum, are sometimes removed from the vaginal walls. They are formed by dilatation of the mucous ducts, as in one of the forms of ranula.

**Injuries** of the vagina are not unfrequent as consequences of pressure of instruments, &c., during labour, when the perinæum, rectum, or bladder may be torn or so bruised as to subsequently suppurate and slough at the spots affected, causing fistulous openings. Clumsy attempts to apply the forceps may lead to perforation of the vagina into the peritoneum. The passage may be wounded by piercing instruments. The breaking of glass syringes may cause severe and even fatal wounds in it.

**Fistular communications** with the bladder, ureters, or rectum, arise through the above-mentioned injuries or else through ulceration. We have a specimen of recto-vaginal fistula caused by syphilitic ulceration ; it is contracting, and such fistulæ may heal spontaneously. But vesico-vaginal fistulæ being constantly irritated by the passage of urine are not cured without a plastic operation.

## UTERUS

**Malformation.**—This organ may be altogether *absent*, as we have now seen on two or three occasions. This may happen when the ovaries, &c., are well developed. On dissection the merest trace of the fundus, as a slight ridge, may be seen on the posterior surface of the bladder, or it may be quite absent ; the organ may be found only in the form of a small sac or small solid mass, or to the small uterus dilated cornua may be appended ; if the ovaries are present, the characters of the female sex appear in the general conformation. Or the uterus may be double, where also there are two perfect vaginæ ; or there may be a disposition to duplicity, which shows itself in the uterus being bicornuate, that is, the fundus separating into two distinct cavities. Sometimes one side of the uterus is ill developed or scarcely developed at all, while the other is well developed. In one case where the left half of the uterus was undeveloped, the ovary and Fallopian tube on that side were absent. This forms the " unicorn " uterus. The divided and the unicorn uterus may produce children, nay, in cases of divided uterus it appears that pregnancy may start on both sides at different times.

To understand the doubleness and the unicorn condition of the uterus we must remember that in the early months of fœtal life the uterus is formed in two distinct symmetrical parts, which subsequently unite and are moulded together to form the single body of the organ. One side fails in the unicorn uterus, and the two sides continue too long separate in the double uterus. The doubleness differs in degree ; sometimes it affects only the fundus, at other times the body, or the neck is implicated. Sometimes through excess of development the cavity is divided by a septum when there is no sign of this outside.

An abnormal figure may be given to this organ from various tumours, &c., compressing it, but especially is it seen altered in cases of ovarian disease, where adhesion takes place and the organ is forcibly drawn upwards ; by this means the cervix becomes immensely lengthened. It is curious to see how deeply it is often imprinted by scybala after death, so deeply that no doubt can be entertained of its being so imprinted during life.

We may here just remark that we may sometimes wish to know, when we open a body, whether the uterus has ever been impregnated, or borne children, or whether it be a *virgin uterus*, as the organ is called which has never held an ovum. The former is somewhat larger than the latter, and the os bears traces of having been dilated, or even lacerated, having an irregular puckered look, as compared with the smooth oval opening of the virgin uterus; the cervix is not so long as in the virgin organ, and the rugæ are in great measure obliterated; and the cavity has a rounded form instead of a triangular shape.

**Malposition.**—*Prolapsus uteri.*—The uterus may descend into the vagina so as to protrude externally and draw down with it the bladder under the pubic arch, compressing the ureters against the arch. We have known on two occasions compression of the ureters from this cause to produce fatal pyelitis. It is comparatively rare for the whole body of the uterus to descend; if it does it generally occurs after parturition, but the uterus may be drawn down by the causes that induce prolapsus of the vagina. What is more common is a prolapsus of the neck of the organ; the body may then descend a little way, but the neck lengthens out enormously, while its lips are somewhat wasted, and the whole may be protruded external to the vulva. The overgrown mass has the general structure of the neck of the uterus, but is more congested; its mucous membrane is generally inflamed, abraded, or ulcerated. The mucous follicles may be enlarged. Sometimes such hypertrophied necks are removed from the uterus by operation, but great care must be taken to avoid opening the peritoneum. Some distinguish as a form of prolapsus a mere elongation of the intravaginal portion of the neck; this is, however, a comparatively unimportant condition.

The uterus may be *anteverted*, or *retroverted*, or *lateroverted;* the whole body being turned forwards, backwards, or to one side. These conditions, especially anteversion, are very common in lesser degrees, for the organ is very movable; they are commonly quite innocent, but they sometimes form a part of the cause of uterine annoyance, especially when the version is complete, so that the fundus of the uterus is downwards and its neck upwards. The bladder, rectum, or ureters may then suffer considerably so as to give rise to serious results. Nevertheless it is remarkable that these versions are so frequently present when no complaint whatever has been made, and we ourselves have never happened to meet with any case where versions of the uterus led to diseased conditions discoverable post mortem. Sometimes the organ is put aside by pressure or traction of tumours or adhesions; here, however, the displacement is evidently of secondary importance.

We sometimes find in the dead body the uterus bent on itself at the os internum, or junction of the cervix with the body; it may either bend forwards and, forming a right angle, rest on the bladder, or, being bent backwards, rest on the rectum; the one condition is called *anteflexion*, and the other *retroflexion;* the former being the more common.

Whatever troubles are traced to these conditions during life, and they are many, they always appear very innocent on the post-mortem table. The same conclusion would follow from M. Panas' observations. It appears that among 114 persons, not suffering from any uterine disorder, 70 had various versions and flexions of the uterus; 40 anteflexions; 12 anteversions; 3 retroflexions; 3 retroversions; and 12 lateroversions.

Dr. Herman subsequently investigated this subject, more especially with reference to the question of misplacements causing menstrual trouble by obstructing the internal channel. It having been more especially stated that anteflexions obstructed the canal, he found this condition not uncommon, but in most cases the interior passage was quite free, and no ill consequences followed the flexion.

The uterus may be drawn up very high into the cavity of the abdomen, when it is fixed to ovarian tumours, &c., the vagina then being elongated and conical in form. Rokitansky says that large fibrous tumours may in their growth tear the body of the organ from its neck.

*Inversion* of the uterus happens after labour; it appears as a red mass projecting into the vagina, and it is difficult sometimes to distinguish from polypus during life, but the absence of the uterine cavity and the sensibility of the surface will generally guide us. It may remain for a long time before its return is effected, and some danger is incurred in rough attempts at its reduction. In an antero-posterior section through an inverted uterus it will be seen how completely the organ is turned inside out into the vagina through the os uteri. The Fallopian tubes and ovaries are drawn into the peritoneal hollow, which makes the inverted cavity of the organ. Death was caused by pyelitis, set up through compression of the ureters by the tumour against the pubic arch. The inversion is not always so complete as in this specimen; the body may be inverted into the neck, or the fundus into the body. Partial inversions are sometimes caused by the traction of polypi within the cavity.

**Congestion and Hæmorrhage.**—In all diseases where the blood is disposed to stagnate—whether from mechanical cause, as in morbus cordis, or from the unhealthy state of the blood itself, as in fevers—we may often find the uterus and ovaries, like other organs, highly congested, and perhaps in a state of catarrhal inflammation; and then, just as there may be a flow of blood from the intestine or other surface, so also there may be from the uterus. We may thus find in fever and other diseases the interior of the uterus containing blood, and the question of menstruation will be suggested to our mind, as it constantly is to the nurses during life, although nothing but a morbid congestion exists. Growths within the uterus lead to intense congestion, and perhaps to hæmorrhage, either by oozing from or ulceration of their surface. We may find a passive form of hæmorrhage into the wall and cavity of the uterus in old age.

**Hæmorrhage about the Uterus.**—*Retro-uterine hæmatocele.*—This name is given to a lodgment of blood in Douglas's pouch. Blood might be expected to be frequently present there, since this pouch is the lowest part of the abdominal cavity; so that it naturally receives the products of any peritoneal hæmorrhage. We have seen some cases in which blood had collected there from such sources as the following: cirrhosis of the liver and heart disease, which had caused intraperitoneal bleeding, though a very rare consequence in those conditions, extra-uterine fœtation, inflammatory congestion of the ovaries from the neighbourhood of ovarian abscess or suppurating ovarian cyst, escape of retained menstrual fluid in imperforate vagina, rupture of a varicose vein in the uterus, operations or injuries about the abdomen, ruptured aneurysm; in short every source of intraperitoneal hæmorrhage. But although any mass of blood so caused is generally adopted as a retro-uterine hæmatocele, something more special to the genital system is usually signified by the term as used by gynæcologists. In particular, a regard is had to its assumed occurrence through escape of menses into the peritoneum, or some other incident causing bleeding during menstrual congestion, or through hæmorrhage from an ovary with its pedicle twisted. There is still much dispute as to the pathology of the tumour-like masses assumed to be blood and found during life behind the uterus, some observers thinking that such menstrual accidents as regurgitation of the menses or rupture of a vein may induce such hæmorrhage in healthy individuals; coagulation and subinflammation causing the blood to form a semi-solid mass. They appeal chiefly to the history of the attack, which often comes as pain and shock in the early days of menstruation. On the other hand, it is contended that we cannot have a tumour from free blood, and that when the blood is encapsulated, as it is found post mortem, there is generally a good deal of disease present, which, probably, may have preceded the bleeding, so that the latter arose from the new vessels of inflammatory false membrane. Here comes in, indeed, the whole question concerning the primary or secondary origin of bleeding and inflammation in serous sacs, which we have considered in the history of hæmatoma of the dura mater, the authority of Virchow bearing in favour of the secondary nature of the bleeding in both cases alike. We have mentioned the rupture of a varicose uterine vein into the peritoneal cavity; this case gives support to the observations of rupture of varicose veins in the broad ligament, &c., collected by Bernutz and Goupil. In the few other examples we have seen, which could be at all referred to proper genital hæmatoceles, it appeared that old disease preceded the hæmorrhage.

Some have described the hæmorrhage as if it had taken place beneath or external to the peritoneum, but it is generally regarded as occurring within the peritoneal cavity arising from rupture of the ovary, Graafian vesicle, or varicose vein, and usually at the menstrual period.

Hæmorrhage into the broad ligament has been described under the name of *peri-uterine hœmatocele.*

**Inflammation.**—Acute metritis, as an idiopathic disease, is rare; it is generally connected with parturition, as we shall presently mention, but it may arise, in the unimpregnated uterus, from operations for polypus, &c.; also we have seen acute metritis and peritonitis caused by cancer of the uterus, perhaps through caustic or astringent applications; it is said to come as an aggravation of chronic catarrhal metritis, but we have never seen this. It is also said that metritis of the fundus causes the painful affection known as "irritable uterus." As a chronic disease it induces hardness of the organ with or without thickening, but this does not show very plainly in the fibrous-looking texture of the uterus. During life there may be a muco-purulent discharge mixed with the more viscid secretion of the cervix.

Inflammation of the mucous surface, or *endometritis*, is believed to be the cause of some forms of dysmenorrhœa, particularly those where at every menstruation a discharge of membrane takes place, as may be seen in our specimens. It is still a question whether these arise as inflammatory exudations or by such an alteration of the mucous membrane as occurs in the formation of the decidua. Dr. John Williams is a strong advocate for the latter theory, thinking it unlikely that the uterus should every month be the subject of an inflammation of a diphtheritic character. It is believed by some that a decidua is formed every month, but instead of breaking up becomes firm and membranous in cases of dysmenorrhœa. The pieces consist mostly of layers of pavement epithelium, but sometimes the membrane has the exact shape of the uterine cavity, hollow with branches corresponding to the cornua, and when microscopically examined in thin sections showing all the constituents of the uterine mucous membrane, loops of vessels running through it and glands being plentiful, while the stroma is a delicate tissue full of spindle- and round-cells, thus showing the characters of a true decidua. Many good authorities have thought that such casts as these never occur except through early abortion, but the answer to this is that they are constantly met with in young girls and virgins.

The post-mortem appearances of endometritis are seen in a softened bloated state of the whole organ from congestion, while in the interior the mucous membrane is red or purple, or subopaque, whitish, or thick, the glands enlarged either singly or in patches. More severe endometritis is found along with the pellicular and ulcerative inflammations of the vagina in fevers, &c.; we then may find the mucous membrane spotted over with follicular ulcers or ragged with more extensive ulcerations; sometimes the uterus is implicated in neighbouring abscesses as of the ovaries, and may be perforated by such an abscess, but this is rare. Sometimes portions of the mucous membrane, in which are, perhaps, several large glands, rise up as a distinct elevations, or even project in a polypoid form, at last forming the mucous polypi of the uterine cavity which become much congested on their surface, and may give rise to great hæmorrhage through the thin dilated state of the vessels on their surface. Of these we shall speak presently.

Grave destructive ulceration (phagedænic) sometimes attacks the neck of the uterus, generally in old or weak subjects.

The inflammatory diseases of the *os uteri* are separate affections, and constitute, according to some obstetricians, a very large part of female disorders connected with vaginal discharges and various uterine disturbances; the lips of the uterus in these cases being said to be frequently ulcerated or abraded, or their follicles in a state resembling acne. More severe abrasions covered with bleeding papillæ are sometimes difficult to distinguish from cancer. These conditions do not constitute fatal disorders, and this is the reason, we suppose, why they are not more constantly met with after death, it being quite the exception to find ulcers in the uterus removed from the dead body; we do, however, meet with less degrees of disease where the *os* is congested or granular, and the epithelium even abraded; conditions which, owing to the turgidity and vascularity existing during life, may then present a much more striking appearance than when seen after death. Syphilitic ulceration is rarely so high up as the neck of the uterus, but it has been met with even within this. Such an occurrence is very rare, however.

Inflammation of the connective tissue about the uterus, *parametritis*, is not unfrequent. Cellulitic and peritoneal abscesses may be formed, and may burst into the vagina, bladder, or rectum.

Gynecologists speak of a *serous perimetritis* or an effusion of serum around the uterus. It has been mistaken for hæmatocele.

The *os uteri* undergoes various changes from labour; it is seen to be puckered or fissured, or even absolutely closed, so that, as a preparation before us shows, the lips of the organ came quite away at a subsequent confinement. It also becomes narrowed by a chronic inflammatory induration of the lips or cervix, and thus arises one cause of painful menstruation, as we see in our specimens. A closure of the *os uteri*, generally the internal lip (*os internum*), may lead to a dilatation of the body of the uterus, as may be observed in some of our preparations. The dilatation arises from retained secretions; these may be purulent, constituting abscess within the uterus or *pyometra*, or they may be blood or menses, constituting *hœmatometra.* If the collection be of long standing it may undergo changes until only a brownish, perhaps fœtid watery fluid is left—*hydrometra.* Such dilatations are rarely of large extent, except in the case of retained menses or "hæmatometra," when the cavity may be widely dilated; but then the stricture is rarely at the os uteri. It is generally lower down the vagina, from congenital malformation, so that the vagina and the neck and body of the uterus are all distended alike, and the os uteri may be difficult to define.

**Morbid Growths.**—*Myoma.*—We have already said, when speaking of the prostate, that new growths within it are of the same structure as that of the organ itself, and that this fact is analogous to what occurs in the uterus. This organ consists of muscular fibre with some connective substance, vessels, &c., but in an unimpregnated state it is scarcely distinguishable by an unpractised eye from simple fibrous tissue. The tumours which grow within it are composed of the same elements, and when the organ is rapidly growing in size during pregnancy, so that new well-formed muscular fibres are becoming developed, then the tumour, if one be present, also increases, and its muscular nature becomes more apparent. The recognition of the essentially muscular nature of the tumours has obtained for them the name of muscular tumour or myoma, which has been substituted for the old name fibrous. The most common and convenient term for them is "uterine fibroid." The fibres are large, translucent, and solid, and appear flat like so many blades of grass. These uterine myomata were once called scirrhus, or, by Baillie, fleshy tubercle; they are exceedingly common, and we are almost daily meeting with them in persons of more advanced age. They are most frequent in the fundus of the organ, and when a section is made they are found embedded in the tissue, forming round circumscribed masses. They are equally dense with or denser than the uterine structure, and the course of the fibres is more manifest; these running in a circular direction, and encompassing a centre from which the disease appears to have sprung. In a large tumour numbers of these concentric masses exist, and the fibres are seen coursing and curling in all directions, but more or less disposed to include certain definite spaces within them; sometimes several small tumours are seen when the uterus is cut through. Those that grow near the peritoneum project from the surface, and then slowly enlarge within the cavity of the abdomen; they are distinguished as the *subperitoneal* and are generally the hardest and most prone to calcareous change; they exist for years, and there is scarcely a limit to the size they may reach, although rarely exceeding the size of the head, as may be observed by examining our numerous specimens. Very often a bunch of them may be seen growing from the surface of the uterus, forming a mass of distinct tumours. At first these are continuous with the rest of the substance, but the connection becomes less as they enlarge, until they at last may become pedunculated, when they may be seen hanging by a slender cord to the fundus, and we believe they have even been found loose in the abdomen, though we have never seen this. They may, however, pass into the broad ligament, and be found there apparently disconnected from the uterine tissue. The firmness of these tumours, or their hardness when cut with the knife, is sufficient to characterise them, and especially if there be any gritty or earthy matter within them, for this never occurs in softer or cancerous growths. Those which arise in the thickness of the uterine wall are called *intramural* or *interstitial*. They may reach a large size, and their growth may divert the course of the uterine cavity and give it a most curious shape. They may be associated with subperitoneal and submucous tumours of the same kind. They are generally not quite so firm as the subperitoneal nor so soft as the submucous tumours; sometimes, but rarely, a portion of such a tumour shows a great development of the blood-vessels, giving it the appearance of *cavernous* tissue. The myomata that form close to the cavity of the uterus constitute an important variety, as they tend to project into the cavity, and so to become "*polypi*." They are rarely multiple, generally softer and more vascular than those in other situations with which they may be associated. They arise in connection with the muscular wall, but as they grow they start away from it, and so may become purely *submucous* and press into the cavity, raising the mucous membrane before them, while the blood-vessels of the latter, and perhaps its glands, become much enlarged. We shall speak of their further history as polypi presently. Occasionally myomata ossify or, rather, calcify, when they become converted into round masses of bone. This change, as we have said, is more frequent in the subperitoneal tumours, but if it occur in a tumour which has grown inwards, forming a polypus (of which we shall presently speak), the uterus may be found filled with what is apparently a large calculus. A specimen of this is on our shelves. We have examined portions of this, and find the structure amorphous, having none of the characters of true bone; for this reason the mistake cannot now occur, which did some years ago, in supposing such a bony mass, which was found in a churchyard, to be an exostosis.

*Cystic myoma* of the uterus, multilocular or "*fibrocystic*" tumour, as it was formerly called, sometimes grows to a very large size, forming a great growth, whose bulk, together with the fluctuation from fluid present in the cysts, may lead to its being mistaken for ovarian dropsy. Under these circumstances the organ forms an enormous mass, in whose tissue are generally several tumours—one, however, being predominant in size. The tumour consists of the usual myomatous fibres, but instead of being homogeneous, its structure is opened up by spaces which contain a watery fluid, perhaps mucoid, and perhaps blood-stained; the spaces themselves are simply fissures or gaps in the tissue and have no proper lining though smooth on the surface. They appear to be formed by a mere widening of the naturally microscopically small interstices in the texture. These cysts may prevail over the solid part of the growth, so that it is largely composed of fluid. They are generally by far most developed in the larger growths, while their earlier production may be well traced in the smaller. The cavity of such a great uterus is often enormously extended. If such a cavity were always permeable by the sound there would be no difficulty in the diagnosis of the disease, but unfortunately for this purpose, the cavity is very apt to be bent out of its course or even occluded by the pressure and traction of the several growths, so that examination by the sound may appear fallaciously to confirm the diagnosis of ovarian tumour.

*Polypus* is the term used for the tumours which grow within the cavity of the womb in a more or less pendulous form. These polypi are mostly of two kinds: the one kind being those myomata which, as we have just described, commence within the uterine walls, and instead of growing outwards project inwards; they constitute the myomatous or fibroid *polypi;* while the others, which form the more common kind, grow from the mucous membrane or submucous tissue, and constitute the *mucous or cellular polypi.* Occasionally a sarcomatous tumour projects in the uterus as a malignant polypus.

In the first form the tumour is hard, though not so hard as those uterine fibroids which grow from the outside of the organ. When enlarged to a considerable size the more superficial parts of the tumour are altered by congestion, and, perhaps, exposure at last, so as to be soft and spongy with dilated vessels; or even to break down into partial cysts or abscesses, or else by simple incompleteness of the formation of the elements they are so soft as to suggest not inaptly the old comparison with boiled udder. These polypi always become more vascular than the external fibroid tumours, but the hæmorrhage which occurs from them during life is mostly from their mucous coverings; in some instances, however, it has been traced to an exposed vessel at the root of the tumour. This fibroid polypus, from commencing within the walls, is at first thoroughly incorporated with them, but in growing it tends to separate itself more from its attachments, and succeeds more or less in this according to its original nearness to the lining. As it grows it expands the uterus. A few of them may form more or less pendulous polypi, or even may be spontaneously ejected or slough away, but generally the continuity with the uterus is broad, and the growth retains its original seat, while the uterus becomes stretched over it until it sometimes reaches the size of the head; the tumour may now begin to slough on the side towards the os, and a part tend to escape, but operative measures being generally unattended with success, and in most cases quite impossible, death occurs from repeated hæmorrhage; or the attempts of the mass to escape may evert the uterus; we find after death the uterus distended to a globular form surrounding the tumour, which is attached to it and intimately blended with it by perhaps half its surface. Still, it appears that sometimes these tumours diminish greatly or even disappear, partly by sloughing, perhaps, but also through a tendency to simple degeneration, which they share with the fibre of the pregnant uterus that they so much resemble.

Fibroid polypi are fortunately less common than those more manageable *mucous polypi*, growing by slender peduncles from the internal surface of the womb which we so often see removed by the obstetric physicians, and with which we are so constantly meeting in the post-mortem room. We see them hanging from the internal surface of the uterus by a peduncle, and their most favourite seat is the *cervix uteri*, more frequently towards the uterine cavity, but some grow near the os externum, and more rarely they come from the fundus of the uterus; sometimes we find them from both together. As soon as they attain any size they pass into the cervix and protrude from the external os. Their attachment is generally quite slender, their form depends on the length they grow to; thus, at first they are nearly sessile and appear then as mere elevations of mucous membrane with one or more enlarged glands, when they are more properly called *mucous polypi.* They afterwards project and droop until they reach the shape of conical eardrops, or else are more globular, especially when they contain cysts, carrying them down some of the submucous tissue, so that their structure is *fibro-cellular.* Sometimes we may see several of them in the same case; thus, the other day we met with four in different stages of development. This kind of polypus differs essentially from the muscular fibroid in being an outgrowth of and from the mucous membrane and submucous tissue; they do not reach a large size. We shall find their surface covered with columnar epithelium. The internal structure is composed, microscopically, of delicate but well-defined fibres, with caudate cells connected with them at intervals, and all embedded in a relatively large quantity of serous or slightly mucoid fluid. But besides these components of the general mass we find not unfrequently that a small polypus has one or more, perhaps a number of, transparent cysts or vesicles on its surface; from the fact of these being sometimes found in the cervix, it has been concluded that they might have been formed from Naboth's glands, which, when very large, as existing in a pregnant uterus, present a somewhat similar appearance. This is probably true, for intermediate stages of growth can often be found, but the cystic development is not limited to Naboth's glands, for we see cysts in polypi from the body of the uterus; and in one specimen the same uterus contains a cystic polypus within the cavity of the body, as well as one in the neck; and in another specimen a similar cystic tumonr, though small, is seen near the fundus. It may be that they are sometimes distinct formations, as in cysto-sarcoma of other organs, but it is probable that they originate in the glandular structures, as Dr. Oldham considered; the intermediate stages we have several times seen, so that, we think, this is most frequently the case. Besides the varieties in the polypi, through the presence or absence of cysts, we meet with very different degrees of vascularity; they give rise to an extent of bleeding and distress very disproportionate to their size.

*Fibrinous polypi.*—Polypi of this kind have created much interest since the discovery of their hæmorrhagic origin by Velpeau and Kiwisch. They are composed of laminated fibrin, and are found in the uterus in cases of previous hæmorrhage, and it is believed by some never without an abortion, while others hold that menstrual hæmorrhage is sufficient to produce them. Some of the recorded cases support the latter belief, and yet there is no doubt that they are generally connected with abortion or delivery. Virchow says he never found them except under these circumstances. In appearance and in the manner of formation they closely resemble the clots found within pulmonary vomicæ in phthisis. They are composed of fibrin laminated concentrically, to the exterior of which ordinary blood-clot may hang in greater or less quantity. In one interesting case we traced such a polypus to a varicose vein when there was no placental sore at all, but the whole mucous membrane healthy, and when besides this ruptured varix another similar uterine varix had ruptured into the peritoneal cavity, so causing death. Otherwise, the few cases we have seen have been after abortion or delivery.

*Sarcoma.*—It is certain that the internal organs may produce sarcoma, and we have now seen several examples of this disease in the uterus. The texture is composed of spindle-cells, and is not so firm as that of simply fibrous tumours; it is softer and of a whiter colour; it does not, however, yield any milky juice like cancer when scraped, and in its mode of growth takes a place midway between a simple polypus and true malignant disease; for without destroying the whole tissue of the organ, like the latter, it springs from a large part of the surface of the uterus, and in its attempts to descend has completely everted the organ; the edges of the growth are sloughing. It would have been quite impracticable in an example like the one we possess to have removed the tumour, and in cases where this has been done it has returned; in one instance, after the existence of the disease for some years, it proved fatal by secondary deposits in the lungs. In another case we met with recently, the disease, which was, as usual, in the body of the uterus, had invaded a neighbouring coil of ileum. But sarcoma of the uterus is a rare disease; the sarcomatous growth is sometimes so hard as to be scarcely distinguishable from uterine fibroid, without the microscope to show its peculiar spindle-cells. At other times it is soft, being of the medullary kind, composed of small spindle- and round-cells, with very little interstitial substance. We have seen cysts within a sarcomatous tumour in one case; also extensive hæmorrhage in another example, in which there were all gradations of hardness and softness from above downwards in the sarcomatous mass which implicated nearly the whole body of the organ. In one case sarcoma coexisted in both ovaries.

*Warty growths.*—Both simple *fibrous papilloma* and *syphilitic condyloma* may be found upon the neck of the uterus. The relation of the larger warty growths of this simpler kind to the epitheliomatous cauliflower excrescence at this part is at least as uncertain as in other situations; we find the forms graduate into each other, and it is probable that in the individual instances a transition from the simpler to the more aggravated growth occurs; but it is very difficult to prove this.

*Syphilitic gumma* is said to have been occasionally found in the uterus, but it is extremely rare; we have never seen it.

*Angioma.*—Virchow describes a single case of small angioma of the uterus.

*Carcinoma.*—This was formerly divided, as elsewhere, into epithelial, scirrhous, and medullary, but now by most authors would be spoken of as sarcoma, adenoma, and carcinoma. Although these varieties do exist, yet the form of cancer which attacks the uterus is remarkably uniform; having the same seat and general characters. The disease commonly begins in the glands of the cervix, causing an induration of its lower part and of the os, and after a certain amount of infiltration a superficial softening takes place, and an open cancerous sore results; this gradually extends upwards and downwards, eating away the tissue of the body of the organ as well as the upper part of the vagina; in bad cases the disease penetrates to the rectum behind, or to the bladder in front, as we see in many of our specimens The patient dies at this time, or even before, from exhaustion, and on post-mortem examination we find, in most cases, that no other part is involved in the the disease, the cancer having been altogether local. What, then, is its nature or degree of malignancy; is it like the more virulent medullary form, or the local epithelial cancer? This can scarcely be answered until the whole subject of cancer is more understood; the general opinion has been very conflicting, tending, perhaps, rather to group it with epithelioma, on account of its local nature, and from death resulting, as a rule, without any other structures being involved than those continuous with the original seat of disease. On the other hand, there is much reason to place it with medullary cancer both because occasionally secondary deposits may be found in the lumbar glands, kidneys,. liver, &c., and especially because the adventitious structure is composed merely of simple cells, and does not generally show those laminated capsules which are peculiar to epithelioma.

According to the more modern views the different growths spring up in special tissues; thus the sarcomatous growth of which we have spoken has its seat in the connective tissue and its cells, whilst carcinoma springs from the epithelium, and is composed of a mass of cells contained in a delicate network. Virchow as is known, would, however, place its origin in the connective tissue, whilst others would find this in the lymphatic vessels. As a matter of fact we find some growths in the uterus of a sarcomatous type, others carcinomatous, and others resembling ordinary epithelioma. The distinction, however, of epithelial from other forms of carcinoma is by no means clear and decided, and uterine cancer we have found to yield more doubtful and intermediate examples than any other kind we have examined.

Mr. Arnott, after careful observation of twenty-two cases, came to the conclusion that epithelial cancer formed about three sevenths of them, and that fewer remote extensions to the ovaries, liver, lungs, &c., occurred in these cases; the rest were almost all medullary, and these infected remote parts more freely. In confirmation of this we have certainly found the uterine cancers which have implicated the ovaries, liver, cranium, &c., to be of a decided medullary kind, but we have not met with well-pronounced forms of ordinary epithelial cancer. The usual appearance shows a meshwork of fibres more or less slender, enclosing masses of cells which approach more to the cells that line mucous follicles than any other. The spaces too are often of regular figure, so that the resemblance to a complex glandular tissue is frequent.

The kind of effect produced in the uterus and parts around varies a good deal. Sometimes the cancer perishes almost as rapidly as it grows, so that there is early a great excavation, the destruction of the tumour being so quick that we may find little more than a great hollow with scarcely a trace of cancer to be seen. The ureters may become implicated, and suppuration then reach the kidneys. We have seen the uterus thus eaten away until only a little piece of the fundus of the size of a shilling remained. The rectum and bladder are in such cases freely eroded. At other times the tumour tissue persists, and we then have seen it block up the neck of the uterus so as to obstruct the exit of secretions, and so produce *pyometra* and purulent inflammation of the uterine sinuses ending in peritonitis. Sometimes the remainder of the uterus is thin and dilated. At yet other times the production of the cancer is so excessive that it forms large masses, which project into the vagina; these, however, are generally prevented from appearing on the post-mortem table by the care of the obstetric surgeon. Beginning in the cervix it kills the patient before the fundus of the organ is affected.

Although in the majority of cases cancer commences at the *cervix*, we sometimes find the disease has begun in the *fundus*, and there is a more rare form of disease where we find the new growth has sprung up all over the surface of the uterus. We have seen the whole mucous membrane covered with a soft carcinomatous growth; and in one case where the growths partook more of a sarcomatous nature, they were nodular on the surface and hung down like polypi; they thus resembled similar growths sometimes seen in the bladder. In the last case we examined the whole surface was shaggy and ulcerated, with polypoid growths here and there hanging down into the interior. The uterus was much dilated, and this with the vagina formed one large cavity. The disease probably begins in the uterine walls, and then grows inwards in the manner described; it is usually diffused over the whole surface, but may be circumscribed. It also frequently involves the walls, and may be found on the exterior of the uterus; it then attacks other organs which become adherent, and its true malignant nature is seen. The uterine appendages and neighbouring intestinal coils partake of the disease. In some cases where the lumbar glands, ovaries, and uterus, have all been involved in one cancerous mass it has been difficult to say in what part the disease commenced. We have seen the body of the uterus perforated, and the peritoneum reached; we have also seen cancer of the uterus associated with colloid.

Dr. John Williams says that, besides the cervix and fundus being the seat of new growths, there is a third spot on the lips or vaginal portion of the uterus, where an epithelial growth may arise and soon afterwards ulcerate. This is the seat of the so-called *cauliflower excrescence.* It resembles the papillary or warty growths of the penis, which we have described, both in its general appearance and minute anatomy. It throws off a watery discharge from its surface, and is found on examination to consist of papillæ containing minute ramifications of arteries covered with epithelium; we have already said that a warty character may belong to an innocent or malignant growth, and as regards that of the uterus we are informed by gynæcologists that it has a basis of epithelial cancer, and that it returns after removal.

**Tubercle.**—It must be a question whether this ever occurs as a primary disease, for it is seldom recognised until after death, when it is found along with other tuberculous conditions as part of a general tuberculosis, and even in those where we have seen it suspected during life the patient has evidently been suffering from phthisis, or tuberculous disease of the abdomen. The malady does not exist as tubercle within the muscular walls of the organ, but is almost entirely limited to the inner surface; thus, as seen in our specimens, the mucous membrane is converted more or less into a yellowish soft granular matter; on attempting to scrape it off it is found intimately blended with the tissue, and no distinct mucous membrane can be detected between the deposit and muscular walls, the membrane having been lost or destroyed in the morbid process. In specimens showing the earlier stages we find distinct tubercles in the mucous membrane scattered about; these break down and form yellow-based ulcers, which extend and multiply until the whole lining membrane of the body of the organ is destroyed, but we find almost invariably that the disease does not affect the cervix, but stops at the os internum, a fact long ago observed by Dr. Oldham. In cases of tubercle of the uterus we, as a rule, find that tuberculous peritonitis is associated with it, and that the ovaries and Fallopian tubes have been especially involved ; the latter have been much diseased and filled with the same scrofulous material, which would lead us to infer that the disease, beginning within the abdomen, has extended to the interior of these tubes, and then has crept down to the uterus ; and so this organ is only affected by the spread of the peritoneal disease.

## UTERUS AND PARTS AROUND AFTER PARTURITION

We shall hereafter speak of puerperal fever, but it may be convenient to mention here the condition of uterus seen after labour. In an ordinary case we find the organ rather soft, and the interior presenting somewhat the appearance of an open wound ; and we may find much blood in it. Otherwise the surface is covered with a reddish-green fluid, and the spot where the placenta was attached is soft, raw, and pulpy. We must remember that the merely raw surface may be easily mistaken for a severe inflammatory patch when it is really in its natural condition. We have found venules open in the placental sore, and blood in the uterus six weeks after delivery. The tissue of the walls of the organ, too, is undergoing changes. We know that, while the uterus is growing so rapidly in pregnancy, new muscular fibres are formed in great numbers. After labour is completed a rapid degeneration goes on ; and if we examine the structure beneath the microscope we find the fibres containing fatty granules showing the degeneration which has already commenced preparatory to their absorption.

In women who die after labour various morbid conditions are found ; in puerperal fever there is often no more local disease than an extreme softness of the uterus, so that the interior can be scraped up into a pulp. But in other cases of the kind we often find more or less grave inflammation of the uterus and its neighbourhood, and it is always a natural question whether, as these are accompanied by pyrexia, the name puerperal fever belongs to them as well as to the cases where the disease is fatal without or with but trifling signs of local complication. As to this question, we must remember that in proportion as the fatal disorder is explicable by local injury and traumatic fever, in such proportion is it of a different kind from fever proper, which begins in a general blood change, and hence we agree with those who draw a distinction between the local traumatic inflammatory *metritis*, *cellulitis*, &c., on the one hand, and the properly *febrile disorder*, puerperal fever, on the other. We have heard Dr. Hicks's reasoning from his experience, which goes to show that much of the puerperal fever arises from exposure to scarlatinal, diphtheritic, and other poisons; there can be no doubt of its intensely contagious nature.

In many fatal puerperal cases, however, we find decided *metritis*, a turbid fluid or even pus being in the uterine wall. It is rare to meet with this throughout the whole of the organ, but it is very common at the seat of the attachment of the placenta or about the injured cervix. Inflammatory products very rarely take the form of abscess in the uterine wall, but we may find small lines or strings of small cysts of pus in the lymphatics, or purulent thrombosis of the veins of the organ. Some authors give great prominence to the lymphatics as the cause of pelvic cellulitis, and even to the production of the white leg or phlegmasia dolens. The mucous membrane of the uterus, as well as its wall, is richly supplied with lymphatics, and these, passing to the exterior, are there thought to set up the inflammation. The peritoneum over the uterus may be inflamed either at the same time as the interior, or, in some cases, without any evidence of the interior partaking. In these cases the Fallopian tubes are often inflamed within so as to contain pus or muco-pus, and it is a question whether the peritonitis is produced by extension of inflammation from the interior of the uterus along the tubes. We have seen *perimetritis*, as the peritoneal inflammation is called, when there was no sign of *endometritis*, and it is possible that the inflammation may extend from the peritoneum into the tubes.

In other cases, where the interior of the uterus is acutely inflamed, the inflammation lights up outside the muscular wall in the subserous connective tissue about the uterus and in the broad ligaments, which become soaked with turbid serum, or suppuration may arise there and extend into the pelvic cellular tissue, constituting *pelvic cellulitis.* Abscesses so formed usually extend upward so as to be opened in the iliac or pubic region, and may rise very high. They more rarely burst into the vagina or rectum. We have seen several deaths by exhaustion when the latter has been the course, whereas recovery is the rule in cases which discharge externally. Such abscesses may find their way into the bladder or perinæum, but they *almost never* burst into the peritoneal cavity. They may be evidently started by direct injury to the contents of the pelvis during delivery; thus they may arise by the suppuration about large blood-clots caused by rupture of vessels. They obtain various names from special writers. The most convenient are those compounded of the name of the organ they are near with the prefix *peri*, such as *peri-cystitis*, *peri-proctitis*, *peri-vaginitis*, *peri-metritis*, but Virchow has used the latter term for the peritoneal inflammation of the uterus, employing the term *para-metritis* for this subserous form.

**Puerperal Thrombosis.**—Three chief conditions favour the formation of thrombus in and about the uterus at parturition; firstly, there is the pressure of it on the iliac veins, &c., tending to induce stasis and coagulation about the valves and circumflex veins within the femoral vein; secondly, there is the greatly dilated state of the veins of the uterine plexuses, inducing stasis; and thirdly, there is the coagulation in the uterine sinuses at the placental sore. There also seems a tendency for the blood to coagulate in the puerperal state, for we see occasionally thrombosis in the arms as well as in the legs after parturition. Either of these three conditions may produce ante-mortem coagulation or thrombus, and the three kinds of such clots are distinguished by Virchow as the *compression-*, *dilatàtion-*, and *placental-* thrombosis respectively. Two dangers of different kinds attend them; first, they become spongy dead masses, which absorb foul materials, either from the sore or from the blood, and when they disintegrate yield them back in a more pernicious form, producing *ichorrhœmia;* and second, they may be dislodged *en masse*, especially from the femoral veins, and be impelled into the pulmonary artery, producing late embolism after delivery. The earlier embolism described by Dr. Playfair and others as thrombosis of the pulmonary artery, we believe to arise as ante-mortem clots in the depression and faintness of parturition, which form in the apices of the ventricles or in the auricular appendices, and are at once dislodged and thrown into the pulmonary artery.

**Rupture of the Uterus.**—This sometimes occurs during the expulsion of the child, and a large rent results, as seen in the numerous specimens in our Museum. The rent, we observe, passes through the os, and extends some way up the walls of the organ, and in most cases runs up obliquely on one side, and sometimes downwards into the vagina. It is generally several inches long, and causes death by hæmorrhage; but sometimes it is less complete, and the bleeding may show itself in the form of a soft tumour in the neighbourhood of the bladder or outer abdominal wall. It may occur in cases of contracted pelvis, or where the uterus is ill-placed for delivery; when labour is unassisted, or when instruments have been clumsily used; or it may be the result of other violence, less well-intentioned; but in most instances, at least this has been the history of our recent cases, there has been no mechanical difficulty, and the subjects have been mothers of several children; and therefore the cause appears due rather to some defect in the tissue of the organ. We do not know, however, that this has been positively proved. There is some difficulty in deciding the question, for, as we have already said, the uterus always shows a degeneration of the muscular tissue at the completion of gestation, and then, even with the microscope, it is hard to say whether this is in excess, or whether it has commenced prematurely in any individual case. We have examined several examples, and in none was there any very marked disease of the tissue. In this strait some adopt a belief that the uterine walls are thin at the part that gives way. Dr. Barnes has pointed out the contraction of the uterus on a dead child as an important cause of rupture.

We have never seen a case of rupture from disease, although we have heard of such an accident from tuberculous affection during the course of gestation, also of a case where there was cancer of the neck of the organ.

The wound of *Cæsarian section* generally gapes widely on the peritoneal aspect.

**Hydatids.**—*Echinococci* have been found in or have escaped from the uterus in several instances, as in an example recorded by Dr. Hicks.

## FALLOPIAN TUBES

**Malformation.**—Both tubes may be absent along with the uterus, or this may happen on one side only, or the tube be but ill developed, the uterus being deficient on the same side. The fenestrated end varies in its complexity, sometimes being little developed, at other times large with two or three openings.

Most of the diseases of these parts are secondary to morbid conditions of the ovary, or to inflammatory processes in the pelvis, or adventitious growths in the abdomen, &c. Thus, they may be enormously lengthened by ovarian cysts; cancer in the lower part of the abdomen may involve them in the disease; and in tuberculous affection of the peritoneum these parts rarely escape; also pelvic cellulitis, arising from whatever causes, generally implicates them.

**Inflammation.**—In cases of pelvic cellulitis, or suppuration about the ovaries, the free extremities of the tubes often become closed and adherent to parts around; and, as the same process implicates their interior, the result is that the uterine ends also close up, so that, thus shut up at each extremity, the tube becomes enormously distended with purulent matter, or else with serum or sero-purulent fluid, producing respectively *hydrosalpinx* and *pyosalpinx.* The results of a more chronic inflammation, as seen in mere adhesions, are constantly met with; the fimbriated extremity of the duct is united to the ovary, and all these parts are much contracted or puckered by the ancient local peritonitis. It was observed by Morgagni that this condition was very commonly found in prostitutes, and was suggestive of a cause of sterility, and our own experience quite confirms the statement. It appears probable that the cause of this condition is usually gonorrhœa, which extends along the Fallopian tubes to reach the neighbourhood of the ovaries, just as it sometimes extends along the spermatic ducts to reach the testes. In one of our cases it coexisted with chancre in the vagina.

The diseased Fallopian tube is so constantly associated with inflammatory products around it that it is very difficult to know in most cases whether the salpingitis constitutes the primary disease or not. The distended tube rarely bursts into the peritoneum, but it sometimes becomes the source of infection, at least we were unable to find any other cause in a case of abscess of the brain, and in another of suppurative pneumonia. Since mere adhesions around the ends of the tubes are included by some, and omitted by others, in their description of disease of these parts, it is difficult to gather from statistics the frequency of salpingitis; thus the report from the London Hospital post-mortem records gives 17 per cent. and that from Guy's 4 per cent.

**Tuberculous Disease.**—When speaking of tubercle of the uterus we noticed the way in which the Fallopian tubes are involved by extension of tuberculous peritonitis ; the tube then becomes distended with a thick, curdy, or scrofulous matter. In early stages the wall can be seen to be sprinkled with miliary ulcers from softening of tubercles in the mucous membrane. This we have seen in two cases. The disease is most severe at the peritoneal end of the tube, and its extension is downwards to the uterus. We have never seen it except in association with tubercle of the peritoneum; but it may be that the extension is sometimes from the tubes as the primary seat of attack to the peritoneum secondarily. When there is much fluid in the peritoneal cavity the thick fimbriated ends of the tubes may stand rigidly up in it, looking like sea anemones. We have met with the disease in children.

**Morbid Growths.**—*Fibrous tumours* rarely form in the tubes, also *Myomata* in them are rare and small.

*Cancer.*—This may arise by contiguity from the tubes being involved in cancer of the abdomen ; we never find them affected by an extension of the disease upwards from the cervix.

*Cysts* may be found hanging from the Fallopian tubes and broad ligaments. These are generally small, not exceeding the size of a cherry or walnut; they are usually regarded as fœtal remains. Thus, one kind of cyst is single, and is frequently present close to the fimbriated edge of the tube; this is traced to the so-called "Müller's thread" on the Wolffian body, representing its dilated end, the rest of it being used for the Fallopian tube itself. Other cysts may be multiple and placed on the broad ligament at some distance from the fimbriated end, that is, in the seat of the "organ of Rosenmüller" or remains of the Wolffian body. Accordingly these cysts are viewed as dilatations of those relics, but a few other cysts yet, sometimes larger, are found at a distance from the organ of Rosenmüller, or even, it is said, within the broad ligament, and these cannot be explained except as new products, as we shall presently mention. Cysts sometimes form in old inflammatory adhesions, and in some cases of the kind papillary growths have been found within the Fallopian tubes.

*Cholesteatoma.*—We have on three occasions met with these small cystic tumours on the broad ligament filled with pearly scales of cholesterine and detritus of epithelium, &c. They are of no clinical importance.

## OVARIES

**Malformation.**—The ovaries may be absent or ill-developed. The failure of development may affect one only, and may coexist with equal imperfection of the rest of the internal genitalia of that side. The ovaries may too, as we have already said, pass into the groin or labia. They may even find their way into the femoral ring.

**Atrophy.**—We sometimes have met with small perfectly smooth ovaries, not one third their natural size, in sterile women. These small ovaries were surrounded by relatively large radiating veins.

It is probably due to the changes which the organs are constantly undergoing that we find the thickened capsule and the puckered fibrous cirrhosed remnant of the ovary so frequently at middle age. It is probable enough that such subinflammatory fibroid states occurring in earlier life may be a cause of sterility; this we cannot say, but it is a subject well worthy of investigation.

**Extravasation of Blood and Corpora Lutea.**—It is very common to find the Graafian vesicles filled with blood; such bleeding is supposed to be connected with a discharge of ova at the menstrual periods. But we meet it too early for this, as at seven years of age; we sometimes find clots, say, of the size of a cherry, associated with a sanguineous fluid in the uterus, in those who have died of congestive disease when not menstruating, as of morbus cordis, or of fevers. When such hæmorrhage as this occurs, the inner of the two walls of the Graafian vesicle thickens a little and grows yellowish, and after the blood has existed in the organ for some time, the usual changes occur in it; it shrinks and becomes brownish and yellowish, so becoming like the *corpora lutea* found after a true conception; hence such patches are called *false corpora lutea.*

The true corpus luteum is found when an ovum has escaped, and its development is proceeding in the womb; a characteristic change then goes on in the ovarium itself: one of the Graafian follicles (or two, if there be twins, for the number corresponds in all animals with the number of fœtuses) is found to be greatly enlarged. When at its full size it is a globular mass of the size of a marble; this, when cut through, is found to consist of a softish yellow matter surrounding a central part of a pinkish colour, the diameter of the latter being about equal to the breadth of the former. What has happened to the follicle is this. The inner of its two membranes has been swollen so as to form a soft, yellowish, vascular substance, which, by the increase of its surface, had to fall into folds to accommodate itself to the follicle. At the same time the outer membrane and ovary around grew more vascular. The thick folded inner membrane reduced the cavity within to a small, irregular cleft, of a whitish colour, and thus what we see on section when the body is fully developed is a yellow substance surrounding a white stellate or zigzag line. On the surface of the ovary, also, a cicatrix is seen. If we examine the section microscopically we shall find a slightly fibrillated material, made up chiefly of fatty, cellular, and fibrillar matter, with a few capillaries; the fat gives it the yellow colour, which is increased by some blood in an altered condition. The body increases until it reaches its full size in the third or fourth month of the fœtus; it then becomes smaller; at the time of natural labour it is much reduced, and in a few months has altogether disappeared. Thus its importance would be only as one amongst the evidences of abortion in the early months of pregnancy. It is distinguished from the false corpus luteum of menstruation by the greater development of the inner folded membrane which causes it to be larger and to project more boldly on the surface.

We may here mention that sometimes blood has collected within an ovary, and so distended it that a *hæmatocele* has been formed. Rokitansky has shown that this is probably caused by a twisting of the pedicle of an already enlarged cystic ovary which strangulates it, and so leads to the bleeding. We have never met with a case, but some examples are recorded. The bursting of such ovarian blood-cysts proves fatal, or the bleeding of a strangulated ovary causes sometimes retro-uterine hæmatocele.

**Inflammation and Abscess.**—Ovaritis may be either a primary or a secondary affection. In many of those cases we meet with in the wards, of local peritonitis in the lower part of the abdomen, connected with some strain or chill during a menstrual period, it is known that a primary inflammation of this organ has occurred. When the ovary is inflamed, the tissue becomes soft and reddish. The Graafian follicles have blood or other inflammatory effusions in them, and pus may be seen in patches or lines, or if the process be less diffuse and grave, an abscess may arise. The exposed state of the ovary within the peritoneum, causing the latter to take on the inflammatory action, renders such acute ovarian inflammation very dangerous, and is one of the causes of acute peritonitis in young women. In these cases the disease is generally limited to one ovary, but if the patient survive and abscess form, the parts get so confounded by the chronic inflammation that it is scarcely possible to trace the disorder to its seat of origin. Also, in connection with parturition and pelvic cellulitis, the ovary may be involved in the suppurating inflammation, if it be not sometimes, indeed, the chief seat of the mischief; in some of those lingering cases of suppuration, in the pelvis and about the genital organs connected with parturition, it has been pretty certain that the ovary has been the starting-point of the disease, although when a number of parts become involved in one extended suppuration, it is difficult to unravel the mass to bring this to the proof. In young people these are probably tubercular.

Both ovaries, nay, indeed, all the internal genitalia, are generally found affected by the time when these puerperal cases come under post-mortem inspection. We have met occasionally with pyæmic abscess of the ovary, and an abscess in phthisis, but no tubercle. We have mentioned that the Fallopian tubes are often bound to the ovaries by false membranes; this is generally in prostitutes, and is traceable probably to gonorrhœa.

We often find the Graafian follicles very thick and large, like boiled pea-skins. The exact consequences of this apparent chronic inflammation of the Graafian follicles we do not know; it occurs equally in multiparous or sterile females, and when the ovary is unadherent we have often noticed the follicles not to be ruptured. The suggestion of Rindfleisch that their thickness enables them to give counterpressue while the follicles dilate to cysts is interesting.

The ovary is very rarely the seat of syphilitic gummatous change; we have never seen it, but the adhesion of the ovaries to parts around is very common in cases of syphilis, but this probably through gonorrhœa having occurred.

**Morbid Growths.**—*Cysts.*—We have already said, when speaking of the kidney and testis, that much difference of opinion exists as to the origin of cysts; this difference of opinion reaches its most distinct opposition in reference to the ovary; the question being whether they be mere expansions of the original Graafian vesicles of the organ, or whether they be new formations. It is probable they arise in both of these ways. In very many cases there can be no doubt that the latter must be the true interpretation, for we find them growing in immense numbers, and forming in the walls of the parent cysts, where there is no trace of proper ovarian tissue, and also springing up on the broad ligament and appendages. On the other hand, we are constantly meeting with ovaria, which, when opened, show Graafian vesicles dilated with blood or watery fluid, and which at once suggest an origin for cystic disease; and, indeed, the ovum itself has been found in cysts of the size of a bean. We have ourselves found an ovum in a cyst half an inch in diameter. Here, no doubt, is one explanation of the frequency of ovarian cysts. Our specimens exhibit these vesicles in various degrees of enlargement.

The division of ovarian cysts into simple and compound, or unilocular and multilocular, as it is sometimes expressed, is a very old one, and to a certain extent strictly pathological. Sometimes, however, the division is artificial only, for an apparently simple cyst is nothing more than one which was originally compound, and the cysts have ruptured into one another. As a rule, however, the multilocular cysts spring up in the ovary proper, whilst the single have originated in the parovarium or other appendages.

*Multilocular cysts.*—As already said, much difference of opinion formerly existed as to the origin of ovarian cysts,—whether they originated in the vesicles, were new growths, or distended spaces in the connective tissue. The latest observers have mostly agreed that their seat is in the Graafian vesicle, or what seems to be a modification of it. In the ovary of infants cysts may be found which seem to be perverted Graafian vesicles, and it is a renewal of this natural tendency to a development of vesicles, which through some abnormal action leads to the formation of cysts. Doran believes that cysts originate in the stroma of the parenchyma of the ovary by its undergoing a retrograde process of development, there being no evidence that they form *de novo* from cells.

We may find a very large cystic tumour, generally unadherent, and with one or two or a few cysts predominating greatly in size, while the rest are much smaller, and at some part or parts there is usually a portion of more solid-looking tissue, seen on closer examination still to be composed of small cysts and representing the remains of the ovary. The capsule is thick, whitish, and fibrous, the interior of the greater cysts pale, and their contents glairy or more tenacious, often turbid and more or less deeply discoloured. The fluid is highly albuminous, of high specific gravity, and contains large cells when examined by the microscope. The inner surface of the large cysts is softish looking and bears more or fewer knobs or excrescences of some form standing up in relief. The walls of the smaller cysts are more vascular, sometimes they have a honeycomb-like or cribriform appearance from fringes of membrane coursing about on them with variable divisions and reunions, while the more solid-looking parts and the section of the solid-looking excrescences look, at first glance, not unlike cancer, being pink, creamy, and vascular.

The appearances under the microscope differ according to the part chosen. The wall of a large cyst is fibrous, generally laminated, the inner part more delicate and areolar with vessels visible in it; the lining covered with epithelial cells, generally cylindrical; these are easily removed. The same cylindrical epithelial cells, perhaps ciliated, generally much defaced, float in the mucous fluid; but pus or blood may be present. If we take the wall of a smaller cyst it shows signs of greater activity, and is more full of vessels and the epithelium more regular. But the most striking characters of this kind of cyst are seen in the formation of bud-like processes within the cysts; they are best brought to view in the most solid-looking parts, and especially within the larger or smaller excrescences on the walls of the principal cysts. These microscopic bud-like knobs grow out and branch, uniting with each other, forming arches, or they extend across the cavity they are in, and thus spaces are enclosed which, in section at least, look very like mucous follicles, being of a tubular form and lined with the cylindrical epithelium, which usually everywhere clothes the buds. They are thus of an adenoid or glandular nature. The buds have vessels in them; they may branch and branch until they no longer are clothed with a distinct epithelium, but the twigs confuse each other and the cells are massed together like those of cancer. This we have seen in parts of a tumour where other parts showed the usual bud, and in this case there were metastatic formations in the liver, thus showing that this form of tumour, when very vivid and rank in its growth, will prove malignant like any other. There is some ambiguity in the interpretation of the appearances, some thinking the small enclosed cyst-like spaces are cysts about to widen, and others that they are rather, as we have just described them, compartments recently enclosed by uniting buds; both views are probably true, so that after the cyst is closed in, as it were, by new growth, it afterwards may dilate and itself bear new buds. Dr. Wilson Fox pretty clearly proved that many cysts were formed by a union of these branches.

*Single cysts.*—Although these may be sometimes met with originating in the ovaria proper, it has been generally admitted of late years that they have a distinct source and are in fact pathologically different from the multilocular. These have again been divided into two kinds, as differing in their characters and origin. According to Sutton and Doran they may be called parovarian and simple.

*Parovarian cysts.*—If the broad ligament be stretched out and held up to the light a number of tubules may be seen within the peritoneum between the ovary and Fallopian tube. These are the remains of the Wolffian body, and the structure with the convoluted ducts is called the parovarium or organ of Rosenmüller. In these tubules cysts may form, and may grow to so large a size as to require removal. They are usually simple, and lined with epithelium which is sometimes ciliated. Growths often occur within them, and if so are not usually of the glandular kind but are papillary, and thus large sprouting cauliflower masses may be seen growing from the interior. These are often branched and sessile, the fluid being watery and not glairy.

*Simple cysts.*—Besides these we meet with large cysts which have had their origin in the peritoneum of the Fallopian tube. These may grow to some size. We may mention also small cysts which are often met with in making post-mortem examinations. They are seen at the end of the Fallopian tube, being connected with the remains of what was once called Müller's duct. They correspond to similar cysts found on the testes and called formerly hydatids of Morgagni. They contain a clear watery fluid, and are lined with a flat epithelium. The fluid they contain is only slightly albuminous, and is of low specific gravity.

We now and then meet with cysts formed by the junction of the Fallopian tube and ovary, and it is very difficult to say in which structure the cyst began. They are called *tubo-ovarian cysts.* A dilatation of the tube is found to communicate with a hollow space in the ovary. The question has been whether accident has brought two cysts together which have then united by rupture of the wall between them, or whether the formation may not have been the result of a perverted physiological process. That is, that after the escape of an ovum the Graafian vesicle became enlarged, and the fimbriated extremity of the tube became permanently adherent over it.

Excepting in a few cases of true cancer, to be mentioned presently, cystic ovarian disease is a local affection, there being no other disease in the body accompanying it; death is caused by exhaustion after repeated tappings, suppuration of the sac, extravasation of blood into it, &c. The tumour may be quite free from adhesions and attached merely by its peduncle; it very often happens, however, that it has formed adhesions to other parts, especially the second form; these adhesions are more frequent in connection with the anterior parietes of the abdomen, if paracentesis has been frequently adopted. The cyst may, however, be attached to the intestines lying behind it, and very frequently the colon is fixed to its upper part by the omentum, which closely covers it. It may thus produce intestinal strangulation. Tubercular ulcer in the intestine may open it. Sometimes also the ureters are found adherent to the cyst, and if not, they are very frequently dilated by the pressure exerted upon them, the dilatation extending to the pelvis of the kidney; the uterus too may have become attached, and if so, being forcibly pulled upwards, is much lengthened, as we have already mentioned; the other ovary is generally healthy, and that it is really so is proved by those cases where, after the extirpation of the diseased organ, the patient has borne children, both male and female; but cases are recorded of an appearance of a second tumour after ovariotomy, requiring a second operation, which, in one instance, was successfully performed. We have specimens on our shelves showing instances where the ovarian cyst has formed communication with the colon, bladder, &c. Sometimes it has ruptured into the peritoneal cavity and the patient recovered.

*Fibroma and myoma.*—Occasionally, though rarely, the ovary may be converted into a very hard solid fibrous tumour; this may contain cysts closely resembling those in cystic myoma of the uterus. In one or two instances they appear to have sprung from the covering of the tunica albuginea.

*Sarcoma—Cysto-sarcoma.*—More frequently these solid tumours are composed of a softer fibrous tissue and are vascular, resembling the uterine polypus. One in the Museum was formerly called fibroplastic, being composed of spindle-cells, and exactly resembling the recurrent fibroid tumours of external parts which are removed by the surgeons; and in another, which has a similar structure, a large cyst is formed, showing the general tendency to cystic formation in these ovarian tumours.

*Carcinoma* is not common as a primary disease; we have never found it reach a great size without cancer being present in other parts of the abdomen. We have often seen the peritoneum partake of the disease, just as it lights up into inflammation in ovaritis, so that cancerous ascites may clinically cover the signs of the ovarian disease that began the mischief. Under these circumstances the growth in the peritoneum partakes of the same nature as that of the ovary, as if it had sprung from germs which had escaped from the ovarian cyst. When ovarian cancer exists as a primary disease it forms, generally, symmetrical masses of opaque whitish nodulated growth, with veins ramifying on their surfaces. The size is rarely large, and the surface is not smooth and shining, like that of ovarian cysts, but more dull and uneven. When cysts form in it, as is not unfrequent, it resembles the cysto-sarcoma, only instead of being composed of spindle-cells, the texture contains nests of rapidly growing epithelial cells of various forms compacted together, with large nuclei and many bright nucleoli, and lodged in meshes of an alveolar tissue. The disease is then eminently malignant, and is propagated to other parts. In several cases of this kind which have occurred here there was generally no suspicion of the disease being malignant except by its rapidly fatal termination within a few months ; and after death the tumour was multilocular, and contained solid growths as in ordinary ovarian disease ; but the liver, lungs, and other parts were filled with cancer. Such a case must be called *cysto-carcinoma.*

Carcinoma of the ovaries is generally secondary to cancer of the uterus or of other parts ; it is usually double. On the other hand, we have seen the ovaries resist a peritoneal cancer extending all around them.

**Dermoid Cysts.**—The cysts known by this name are characterised by containing hair, as well as generally fatty matter, with sometimes teeth and amorphous pieces of bone. They have no connection with the class of diseases of which we have been speaking, and yet we have met with them sometimes, as it were, casually associated with simple cysts and with malignant disease. In a very remarkable case recorded by Mr. Hulke there were numerous secondary dermoid cysts about the liver ; the primary disease being cancer associated with dermoid cyst about the ovary ; also in a case by the late Mr. Moore there were many small dermoid cysts in the omentum, &c., near a large one. So that it appears they may prove infectious, at least like cartilage, when associated with malignant growths. These cysts are generally met with accidentally in persons of all ages, even in childhood, who have died of other disorders ; although, indeed, in a case that occurred lately one of them was the cause of death by rupture and subsequent peritonitis ; they are not generally larger than the distended bladder, if so large. The most frequent contents are white fatty matter and hair. The former is no doubt secreted by the sebaceous follicles found in the lining membrane of the cyst, and is fluid while the patient is alive, but becomes solid as the body cools; so that it may be seen in our bottles in the form of a number of perfectly round balls. This has been proved in the case where the body was still warm when opened; the fluid flowing from the cyst solidified on the table like a layer of tallow. It entirely dissolves in ether, leaving no membranous residue. The hair is in bundles, and is generally, we believe, of a light colour; when examined it is found to be pointed at both ends, but this probably is an acquired condition, for no doubt it has been formed on the surface of the cyst, and thrown off. In the specimen which occurred here lately, there is a good cuticular lining, and from it a number of hairs are growing; a section of the skin shows well-formed hairs in their follicles, and a very rich supply of sebaceous glands, which were no doubt constantly pouring out their fat. In another specimen there were also three teeth embedded in a piece of bone, which Mr. Salter thinks have been there many years, although they are the primary or milk teeth, and, what is remarkable, he thinks the teeth contain in their pulp true nerve substance. In the Warren Anatomical Museum are two bones from such a cyst, with seventeen and thirteen teeth in them respectively. In a case of Graily Hewitt's the envelope contained muscular fibres.

We shall not deal exhaustively with the various opinions given as to the origin of these remarkable tumours, containing within them the parts necessary for the construction of a fœtus, but we may mention some theories raised to account for them. First, one founded on Owen's view of "*parthenogenesis*," in allusion to those kinds of insects who, from one impregnation, continue to bring forth successive generations; suggesting that in the human body, in an exceptional case, the fœtus when born of its mother might contain within it the germ of another fœtus. A second theory is that which assumes an *included ovum;* where, after the impregnation of the ova, one shall envelop the other wholly or partly, and thus one shall be found projecting from the other. In these cases, however, the included ovum is either a heterogeneous mass, or shows merely a resemblance to a fœtus, and may be met with in any part of the body. It has also been thought that they spring up in the ovary as an independent act on its part as a *generative* organ, but dermoid cysts are not peculiar to the ovary; they occur in other parts, even the brain and its membranes, and, as we have seen, may be repeated by malignant infection, so that it appears they spring up in the ovary in the same way as elsewhere, and as obscurely as other tumours.

Lawson Tait has described extra-peritoneal cysts, which were thought to be ovarian, but were really formed between the peritoneum and abdominal walls. He believed they originated in the remnants of the urachus. They usually contain purulent fluid, and in one case urine was also present. Mr. Bland Sutton, in confirmation of their origin, says that *allantoic cysts* are met with occasionally in the dead body, both of human beings and animals. They lie between the peritoneum and the sheath of the rectus muscle.

## MAMMA

**Hypertrophy.**—The natural increase of the gland in lactation is one of the most remarkable examples of hypertrophy which could be instanced. Sometimes the breast grows to such an enormous size that its removal is required, and this without the formation of a distinct tumour in it, but as a uniform increase of the whole organ. This is very rare; in one case of the kind the organ, when excised, weighed many pounds.

**Atrophy.**—In the decline of life the mammary gland atrophies, and in some old women, from a loss of fat and shrinking of the secreting structure, the remaining fibrous tissue has formed a hard lump, which might be mistaken for true scirrhus; at least we have been questioned as to its being of such a nature more than once in the post-mortem room.

**Inflammation and Abscess.**—Superficial excoriations and fissures about the nipples form a very painful affection during lactation; sometimes they lead to erythematous inflammation. The skin about the nipples is liable to molluscum contagiosum; also to syphilitic ulceration and condyloma. The deeper inflammations we frequently meet with during lactation, and more rarely as idiopathic affections, or resulting from injury.

The inflammation may be either within the mammary gland or in the tissue around or beneath it. The tendency is to form abscesses which may be localised in some part of the gland or more diffused around it externally. They rarely excite pleurisy. They are generally opened by the surgeon; sometimes they burst in two or three places, and lead to a formation of fistulous canals. Some of these abscesses become chronic, and have been known to dry up, leaving calcareous relics.

**Morbid Growths.**—The female breast is frequently affected with tumours, and these are often complex, and differ much from each other in several distinct ways. Thus, they differ in some dilating the ducts into cysts, while others do not do so; and, again, they differ in being composed of different materials. The cystic dilatations are very striking to the naked eye, especially when pedunculated masses of the tumour of various forms grow into these cysts; but we find cystic tumours with very much the same appearance, composed of textures essentially different from each other; thus we may get cystic adenoma, fibroma, sarcoma, or myxoma, looking so much alike to the naked eye, that the microscope is required to distinguish them; and, again, we may find adenoma, fibroma, myxoma, &c., free from cysts, but essentially of the same nature as if these had been present.

A few years ago, when Mr. Birkett proved that many of the intracystic growths are composed of a structure identical with that of the mamma, *i. e.* were "adenoceles," the interest of this discovery caused it to prevail in the consideration of all intracystic growths; and as most of these growths contain some glandular elements, such elements were regarded as the predominant feature in the tumour, and hence it came about that these intracystic tumours were all called adenocele more or less confusedly, and the old term for them, "cysto-sarcoma," fell into disrepute. Mr. Birkett's own plan of teaching was then to show to his class how the various forms of these tumours resemble the artificial dissections of the mammary gland, according as more or less of this uniting fibre tissue is taken away. Thus, a section of a female breast shows merely a uniform fibrous surface; but if by dissection some of the areolar tissue be removed, it is found to be separable into lobes; these lobes, again, by further dissection, may be separated into lobules; and these again, by a yet further removal of connective tissue, into the primitive acini or cæcal terminations of the ducts. Corresponding to all these degrees of dissection, so may the new tissues be developed in the adenoceles: in one case the gland tissue is at once seen by a magnifying glass, minute lobules being connected by ducts; in another these are clustered to form larger lobules, which are distinctly seen by the naked eye when the tumour is opened; and in another case the connective tissue so preponderates that the whole is conglomerated into one uniform mass. All these varieties may be seen on our shelves, some of which, growing many years, reached several pounds in weight, and have very uniform solid structure, while others display the lobules of various sizes. The preparations were chiefly made by Mr. Birkett's admirable dissections, and at the time when he taught these views they were in accordance with all that was known of the connective tissues spoken of as interstitial between the glandular acini.

But since that time the microscope has shown that the material in which the adenoid elements in the growths are embedded is itself of too great importance and too various to be ignored. So that while Mr. Birkett's teaching as to the existence of glandular elements remains as true as ever, it is important to know that along with these elements others of a fibrous, sarcomatous, or myxomatous kind are apt to be present, and may greatly prevail and determine the character of the whole growth, so that the interstitial material comes to have a new consequence, and the question arises often, when the gland substance is in smaller amount, whether this may not be altogether old and represent included portions of the original gland. All these conditions have been carefully studied by Mr. Birkett, and may be found fully described in his work on the breast.

The recognition of these connective elements of intracystic growths is carried to an exclusive extreme by some recent writers, as Rindfleisch. He teaches that the tumours with cysts into which growths project are of a totally different class from adenoid. So that in his main division, viz., into mammary tumours from epithelial growth, and mammary tumours from growth of connective tissue, he puts adenoid tumours in the first and cysto-sarcoma in the second, thus separating them entirely. We think that this view is wrong in its exclusiveness, for, we do find typical glandular tissue forming growths into cysts, and this in a large number of cases and to a large extent. This fact he appears to ignore altogether. On the other hand, it is necessary to admit that the elements of these growths, which give them their great importance, are often the interstitial connective substances, and these may exist in such intracystic growths without any glandular substance at all, and they are various in their kind.

Another point much confused at present by histological anthorities is the relation of the old adenocele or chronic mammary tumour, now called adenoma, to carcinoma. We will endeavour to show this confusion, for it must be avoided rather than cleared up by the student. The definition of carcinoma is not very different from that of adenoma; indeed, it is scarcely possible to draw the distinction without going into details about very minute points, especially the characters of the cells, which are large and have large nuclei in carcinoma. Formerly these cells would have been thought to make a very sufficient criterion, but Virchow repudiated the "cancer-cell," and very ably directed attention to the plan of histological composition of tumours as promising a more sure means whereby their nature could be determined, and since then the cancer-cell has fallen into disrepute. But although there is no doubt that a cancerous action does belong to tumours, such as lymphoma and some sarcomas, which have no "cancer-cells," yet in this particular question between adenoma and carcinoma it is very important to note that the cells of the latter are very different from those of the former, so that as between these two kinds of tumours the cells do form important distinctions, however comparatively unimportant they may be elsewhere; indeed, it is difficult to draw any histological distinction between these kinds without putting the cancer-cells in a prominent place in it. The structure of the gland when seen in section, shows its acini cut across, so that we see spaces lined with epithelial cells, the spaces themselves having proper walls of fibroid stroma, which form a meshwork in the section, defining the outlines of the spaces containing the epithelium; in whose walls a good observer will detect capillaries and lymphatic channels. Now, the structure of a cancer would in description correspond very much with all this; we have epithelioid cells in the interior of spaces which are separated from each other by a meshwork of fibroid tissue. Hence, if we take up the relations of the two with a philosophical desire to see analogies and keep in view the generic common likeness, we may easily look on adenoma and carcinoma as in one category, or essentially the same thing, as indeed Rindfleisch does, but it is a serious practical misfortune if we are obliged to weaken the distinction between innocent and malignant tumours; a practical aim was before those who commenced the study. It was not to reach an abstract knowledge of tumours, but to find new means of discerning their clinical tendencies that the microscope was called in aid.

The difference between adenoma and carcinoma is commonly the difference between life and death, and as the microscope has latterly plainly tended to confuse these kinds of tumour, we shall not in our descriptions of them recommend at all an exclusively histological view, but rather consider them in a general way, using all the means which our senses furnish towards knowing the several kinds.

We shall describe the tumours of the breast as—1st, simple cysts due to dilatation of the ducts; 2nd, solid growths of structure similar to that of the mamma, or growths of sarcoma and myxoma mingled more or less with mammary structure; 3rd, tumours of the same composition as the last with cysts into which often project polypoid masses of the tumour of various shapes; 4th, carcinoma in its several forms; 5th, other occasional tumours, as certain large cysts, enchondroma, hydatids, &c.

*Cystic dilatation of ducts, or galactocele.*—Sometimes one of the ducts dilates, and the milk collects until a large tumour is formed; when opened, the fluid escapes, or, as in a specimen of Mr. Birkett's, a change may take place in it, whereby the fluid parts are absorbed, and the solid mass of casein remaining is found in the cavity. There may also be a general dilatation and disease of many ducts, as shown in some of our specimens.

*Adenoma—Chronic mammary tumour.*—By this we mean a tumour resembling the natural tissue of the breast. This is not frequent in a pure form, but it is found usually combined with sarcomatous or myxomatous substance. It arises in one or several lobes of the gland, sometimes so superficially that the whole gland appears to be free though pushed aside or spread over the tumour, which may be lobed, or nearly globular. There is a capsule composed of connective tissue compressed by the tumour. Section shows a subpellucid succulent mass almost homogeneous, and so differing from the more dangerous sarcoma and myxoma, which have a conglomerate, discoloured appearance.

The tissue, examined by the microscope, is not so perfect as that of the normal breast, for the larger ducts are mostly wanting, it being very rare to find any fresh formation of them, but their cæcal terminations are seen as in the healthy gland, and lined with epithelium in the same manner; it is these we must look for when we are examining a suspected specimen by the microscope, and we shall soon discover the rounded terminations as we see the healthy gland drawn in our books.

If expert in the use of the instrument, we can generally see the blood-vessels and lymphatic spaces between the acini, and we shall see that the glandular epithelium is just like ordinary mammary glandular epithelium in the active gland. Also, generally, the lumen of the follicle is preserved (we have repeatedly seen this in well-marked adenomatous tumours, although we have heard it denied). The differences from cancer which such tumours show, sum up, perhaps, best in this, that these mammary tumours are exactly like active mammary gland and cancer is not. The microscope brings out the minuter differences as the naked eye shows the larger. In carcinoma we do not see with the microscope well-defined acini with regular vessels in their walls and proper glandular epithelium lining their cavities, as we do in these growths; also by the naked eye the following appearances distinguish them from cancers; their defined surface enclosed in a capsule of compressed tissue, their structure pellucid and homogeneous, the substance not retracting on section, no milky juice yielded when the surface is scraped, and no infection of parts around or of the lymphatic glands.

*Adeno-sarcoma—Adeno-myxoma.*—We have just described adenoma as a development of new glandular substance in the form of a tumour. Such new glandular substance includes several components, in particular the acini, the interstitial tissue between them, blood-vessels, and lymphatics; now in some cases masses of new growth appear in the breast, which when we examine we find to contain a variable proportion of glandular acini with their characteristic epithelium, but the interstitial substance between these acini is in great excess, and instead of being simple connective tissue, has the structure of sarcoma—that is, it is composed of spindle- or round-cells with intercellular substance. When we see this structure under the microscope and find a few glandular elements and much sarcomatous interval, the whole arising from a lobe or two of the original gland, or perhaps from a larger part of it, the question naturally arises whether the acini are not persistent elements of the normal gland and the sarcoma the only new matter. When the proportion of gland is small this is probably the case, but we have seen several examples wherein along with sarcoma there was an evident excess or hypertrophy of the glandular acini. On the other hand, no doubt can exist that in many cases the acini represent only persistence of the natural elements. Such growths are called *adeno-sarcoma.*

Similarly we find other cases in which the like compounding of gland tissue with myxomatous structure occurs in varying proportions. Such tumours are called *adeno-myxoma.* The appearance to the naked eye in such tumours is not unlike that in simple adenoma, but they do not show the glandular appearance, being more uniform and more opaque. Generally, too, the tumour is more divided into lobes, and the lobes themselves differ in hue and consistencey. The glutinous touch and gelatinous consistence of the myxomatous form distinguish it from sarcoma.

*Sarcoma.*—In some cases we find large tumours of characteristic sarcomatous structure in the breast—Paget's recurrent fibroid, the composition of these being quite typical.

They are more distinctly fleshy and subopaque than adeno-sarcoma, but the microscope is required to distinguish with certainty between them. These tumours are likely to recur after removal, as in some of our specimens; the subsequent growths are generally softer than the first, and it has been said that a change of type appeared after frequent recurrence, so that the structure was like carcinoma; but this may have been alveolar sarcoma.

*Myxoma.*—Likewise in some cases myxoma arises in the breast, quite typical in its character. It may be found in the form of a distinct pendulous tumour, or an encapsulated growth, pushing the breast aside or including much of the mamma. In section a myxomatous tumour generally appears composed of lobes of different consistence and colour, but all more or less gelatinous. We can often trace all degrees of softening, to the formation of cysts. At other times the consistence is fleshy. We have met with examples in which whitish richly cellular material surrounded the blood-vessels for some little distance, being then enclosed by a defined surface, so that the blood-vessels formed thick, white, wormlike coils in a clearer and less consistent material, such as we have described under the name of peri-angioma. Soft myxomata of the breast are likely to recur.

*Fibroma.*—Tumours of well-developed fibrous tissue occur sometimes in the breast, and may reach a large size. These are innocent, and form rounded, movable, massive growths. They must not be confused with atrophic carcinoma, which sometimes shows little more than fibrous tissue.

*Cystic adenoma—Cystic sarcoma, Cystic myxoma, &c.*, are a set of terms which express the simple fact that cysts may develop in any of the forms of tumour to which allusion is made in the name. Such cysts are at least of two distinct kinds. Sometimes they are dilated ducts; sometimes they are formed by a solution of the tissue; perhaps they also arise in other ways, as by hæmorrhage into the soft mass, by widening of casual interstitial spaces through accumulation of fluid, &c. The cysts caused by dilatation of the ducts are generally not far from the nipple. A duct may be traceable into one or other of them. Further, the microscope shows that they are lined by cylindrical epithelium. Such cysts are often of large size and numerous, but they are usually flattened into fissures, and on stretching them open we find spaces into which polypoid, laminated, or cauliflower-like masses project from the wall. They are usually described as arising by obstruction and distension of the ducts. From this we should expect them to be hollow spaces instead of fissures, as we have described. To explain their being closed, and yet due to distension it is generally supposed that they once were uniformly distended, but that the polypoid masses grew into and closed them; there may be some truth in this, but we believe that the assumption of their ever being dilated into wide spaces and subsequently filled up is incorrect. We may remember the enormous surface which the prostatic urethra shows in an hypertrophied prostate, forming a fissure of many square inches superficies. This is caused by the growth of the superficial urethral tissues, which share in the formative activity, while they retain connection with the growing mass of the prostate, and so while they increase are drawn and thrust into such shapes as the new developments may compel by their pressure and traction. In a similar way, partly by growth of the duct-surface and partly by its being drawn and thrust by the irregular pressure of the unequally but greatly enlarging tumour tissues around, the surface of the duct is expanded and stretched about, around, between, and otherwise among the ever-increasing lobe-like masses which began in its walls, until it becomes a great irregular fissure amongst these lobes, such that when we stretch the fissure they appear to have grown into a space. This is, we believe, the mode in which the fissure-like cysts arise; they never were hollow spaces, but grew as chinks and clefts. They are often very numerous, so that the section of such a tumour has been well compared to the cross cut of the heart of a cabbage. If we inquire why in some cases the ducts should thus form cysts and in others should not do so, we discover, in answer, a certain analogy in the behaviour of the surface of the skin and mucous membranes in the formation of tubers, condylomata, and papillomata; for in tubers which form in the deeper texture, the surface of the skin grows little; in condylomata, which form in more superficial texture, the surface becomes complicated, and in papillomata, which form from the very surface, it becomes richly and intricately complex, thus showing that when tumour growth implicates a natural tegument close to the surface there is no limit to the extension of that surface which it may bring about. If, then, we suppose that in these cystic growths the cause of the tumour implicates the surface of the ducts, expanding it like papilloma does the skin, while it still retains its epithelial surface, we can understand how such cysts may arise when in other cases the surface of the duct not being implicated, no such expansion of the duct occurs. As we have already said, such polypoid development in chinks and fissures occurs both in adenoma, sarcoma, myxoma, and intermediate forms, giving rise to cystic varieties of those forms.

Besides the cysts thus developed from the ducts, we meet with others plainly produced by solution of the tumour tissue, especially in myxoma. Hæmorrhagic cysts are more frequent in carcinomatous and other malignant tumours; besides these we occasionally meet with small globular serous cysts, apparently caused by distension of the glandular acini or of interstitial spaces. Such cysts may be found in the mamma without any tumour, or, when a tumour is present, they may be at some distance from that tumour. Mr. Bryant says in old and obsolete glands these cysts are not uncommon, and he has called the disease "cystic degeneration." The cysts are small and contain a dark glairy fluid, and have probably arisen by dilatation of the lactiferous ducts.

**Carcinoma.**—As we have before said, this form of disease depends mainly for its physical characters on those of the organ wherein it is developed, and so it follows that, in a part which, like the mamma, contains much fibrous tissue, the cancer is of the scirrhous kind; and it is most probable that its hardness is due merely to the fibrous network belonging to the original structure of the gland itself. Thus it is that if a soft or encephaloid cancer of the breast be met with, it is only when the tumour has grown to an enormous size, so that the new soft cellular elements greatly preponderate, or else when a secondary tumour has arisen after excision of the breast, so that the growth has no old fibrous tissue in it, and the stroma or matrix of the growth is a new formation, and is of a comparatively slight kind. In these secondary growths, however, the tumours are at their commencement very hard, since they spring up in the skin around the cicatrix, and this consists of a dense fibrous structure. In ordinary *carcinoma* of the breast the tumour is hard, and the cut surface shows a fibrous-looking fabric.

The leading characters of such a cancer are, first, its tendency to contract, exerting tension on the parts around, drawing in the nipple and skin in a puckered way, and in section showing tough fibrous cords running into the fat around. The same internal tension is shown by the shallow cupping of the surface on section, which is a remarkable feature. The next leading character is the tendency of the elements to degenerate, shown by the whitish, opaque, curdy, or even cheesy patches, minutely mottling the surface, which elsewhere is opaque but creamy or milky with a pinkish cast, the whole minutely reticulated with faintly bluish lines of fibrous texture. Again, other characters are the implication of the glands and the continuous infection of the skin and other tissues, so that they are incorporated with and not merely stretched over the tumour.

Carcinoma is distinguished into several kinds according to the character and quantity of the cellular element. Firstly, *medullary*, where the cellular growth is free and the stroma therefore proportionately soft. This kind is liable to hæmorrhage and degeneration; it is rare in the breast, and may grow to a large size. Secondly, carcinoma *simplex* is the ordinary form; its characters are those above described, the consistence being about that of a raw potato. Thirdly, scirrhus or *atrophic* cancer shows a great proportion of the fibrous substance. The nests of the cells being very few, and often being in a state of degeneration, which may be so complete that the whole looks like fibrous tissue, give rise to accounts of fibrous cancer. Some distinguish between scirrhus and atrophic cancer; examples with small cells and no fatty degeneration, but with a great preponderance of fibre being called by the former name, while the degenerating forms are called atrophic. A *tubular* variety is spoken of by Billroth in which the cells run in tubular spaces thought to be lymphatics.

The original starting-point of the disease is a question on which opinions are extremely various. Virchow considered its malignancy to depend on a growth of epithelial elements from connective corpuscles. The "*heterologous*" character of such a development on his view would explain the malignancy. The older opinion of Rokitansky, which still has distinguished expounders such as Thiersch, is that the epithelial elements are developments of the natural glandular epithelium, Rokitansky thinking this to be through the local influence of the glandular neighbourhood upon fluids effused, while Thiersch considers it an hypertrophic solid outgrowth of the normal gland. Rindfleisch adopts an intermediate position, and describes the gland-follicles as at first proliferating into cancer, and afterwards affecting the interstitial substance with the action; at the same time he leans a good deal to a rather distinct view especially favoured by Köster and Billroth, in which it is thought that a principal seat of the activity of carcinoma is the endothelium of the lymphatics. Thus we see that the prevailing solidism gives a start in life to abundant conflicting theories. If any other tissue were present no doubt some one would soon advance it as the origin of cancer. According to our own observations all the tissues are alike implicated, but the earliest stage appears to be the formation of great groups of cells in the place of individual connective-tissue corpuscles. Still there can be no doubt that in the further progress of the cancerous change the epithelial elements are implicated. Close around the cancer we often see crowds of small corpuscles; some think these are young cancer-cells, but they may be due to inflammatory infiltration. For the sake of classification a simple order is sometimes made, as *sarcoma* of fibrous or medullary kind as spindle-cells or round-cells prevail; then adenoma and carcinoma, both originating in the glandular constituents, the former exhibiting an increase of lacteal structure, whilst the latter shows merely a multiplication of epithelial cells. One, however, is benign and the other malignant. There is no practical division, however, of this kind as the various new tissue growths become intermingled.

Mr. Rushton Parker showed, at the Pathological Society, a case of adenoid tumour of the breast which had on its surface a growth presenting all the features of cancer. This was confirmed by the "Morbid Growths Committee."

We sometimes meet with cases where the tumour is evidently cancer but containing cysts in which growths have taken place. The section gives out a milky juice and the glands are infiltrated, whilst the cysts or dilated tubes contain dendritic growths covered with polygonal cells. These growths, originating in ducts, might warrant the name of epithelioma but the alveolar nature of the tissue with its large cells assimilate it to cancer. This form of disease is probably the same as that described by Cornil and Ranvier as *villous cancer*.

Examples of it are met with in that form of malignant disease of the mamma which is associated with a cutaneous affection of the nipple. Paget was the first to describe *carcinoma of the breast following eczema of the nipple*, and afterwards he was followed by Morris and Butlin. Thin, however, made the most careful study of the disease and came to the conclusion that the affection of the skin was not primary but that there was a previous disease in the epithelium of the ducts. The affection of the skin therefore was not simple eczema or ulceration but a "malignant papillary dermatitis of the nipple." The disease began as a growth from the epithelium of the lactiferous ducts of a columnar character, and therefore more of the character of adenoid than ordinary scirrhus. Thin therefore proposed to call it "duct cancer," but it seems to be of the character just spoken of as villous cancer. This author then regards it as the primary affection, which, involving the neighbouring connective tissue, sets up the ulceration of the skin. Paget thought that the long-continued irritation of the skin at last resulted in a malignant action as he instanced the occurrence in other parts of the body. Morris showed the true malignant or cancerous nature of the disease by observing the secondary growths in lung, liver, and other viscera.

Cancerous tumours may remain for many years without causing inconvenience. In such cases the disease is of the atrophic or scirrhous kind. Sometimes, but very rarely, the cancer has been known to undergo calcareous change. In a few cases cancer has been found associated with a great cyst, the cancer forming only a patch on one side of it. Such cases have been of slow development. Sometimes cancers do not return after removal, but in most instances the whole history endures for only a year or two ; the disease returns on removal, and after death the internal organs are commonly found affected ; mostly the liver, lungs, and not unfrequently the bones of the skull and spine with dura mater. The disease may be propagated through the body as independent deposits, but very often the interior is affected by a gradual extension of the cancer through the walls of the chest to the pleura, or along the cervical to the mediastinal glands, thus reaching the interior of the body : on several occasions, however, we have examined persons where the local disease has caused death by exhaustion, and no cancer was found internally, although the organs may have been otherwise degenerated. As we have before said, the general atrophy of the system so often associated with cancer is due immediately to interference with the function of some important organ, and thus it is that in these cases of cancer of the breast the bodies are often very fat, affording a strong contrast to the wasted state of those dead from cancer of the stomach.

*Colloid carcinoma.*—This is not frequent, yet not very rare. It usually appears as a colloid transformation of the cells of a carcinoma with great enlargement of the containing spaces until the reticulum of the cancer is reduced to fine threads, and the cells to a gluey material containing a few corpuscles. Some examples called coloid have proved on examination to be glutinous and soft myxomata.

*Enchondroma*, in a few instances, has been met with in the breast. It is seen in the form of small glistening nodules and is met with in both innocent and malignant tumours. Rarely bone is met with.

*Lipoma* may occur about the gland, and simulate tumours within it.

*Hydatid.*—We need not explain that the hydatid disease of the breast, spoken of by older writers, is what we know now as cystosarcoma, and that real hydatid is a very rare disease. We have seen, however, a few such cases ; in one specimen we see the parent hydatid or ecchinococcus, filled with smaller ones, and in another a cysticercus.

## UTERO-GESTATION

We shall not say much on this subject, as it is so fully treated of in works on obstetrics ; but there are certain parts of it which are constantly coming before the morbid anatomist, and upon which his department has thrown great light ; for example, abortions, which formerly were thought to arise from some accidental circumstance, are now known to arise, in the majority of instances, from disease of the ovum.

## OVUM

We have repeatedly said that most of the morbid actions in the body are chronic, and the same statement applies to abortion. It was formerly thought that this was always due to some violent exertion or excitement on the part of the mother, which caused the uterus to discharge its contents prematurely ; but an examination of the aborted ovum generally shows a change in it, which must have been progressing for a considerable period. In some cases it is probable that from the time of conception the ovum is so slightly blighted that no embryo appears, while yet its membranes may continue to grow. We find all degrees and varieties of imperfection of the embryo in different cases.

We often, however, have ova shown us which appear healthy ; when this is so the deciduous membrane is perhaps seen on the surface, but it may be retained and cast off afterwards, so that the chorion then forms the outside of the aborted ovum ; or a mass resembling a mere blood-clot may be discharged, its separation being due to sanguineous effusion in the deciduous membrane, which has caused a separation of the chorion, and thus the ovum may be found in the clot. At a later period the cause of abortion will generally be found in a *disease of the* *chorion;* for when this is gravely affected, containing as it does the vascular loops through which the nourishment is conveyed from the mother to the fœtus, the ovum necessarily drops off. That the disease is due in the first place to some constitutional defect on the part of the mother is suggested by the repeated occurrence of abortion in the same patient; no doubt a chronic inflammatory state of the interior of the uterus would be sufficient to cause it. When the chorion is examined under these circumstances its villi are found to be of a white colour; if some of them are placed under the microscope they appear quite opaque from the presence of crowded fat-globules and granules, the natural cellular structure having disappeared. In some of these cases also we find the villi dilated into small cysts, approaching in character the form of disease we shall next describe. These changes may be found in the chorion at a very early period. Thus, we have in two or three instances found a spherical mass of the diameter of a florin, which was quite white on its villous surface, while the interior was much discoloured with hæmatoidin pigment, no trace of the embryo being discoverable. Such cases are not easy to understand; they suggest that some bleeding occurred very early in the formation of the embryo, stopping its development, the ovum being retained until the embryo wasted away. In one of these cases the villi of the chorion showed commencing cystic degeneration—a fact that favours the belief that the cystic chorion develops after the death of the embryo.

In most aborted ova of three months the membranes are firmly united and blood is extravasated into their substance, all trace of embryo being wanting. Dr. M. Duncan describes an unequal growth of chorion causing abortion and Dr. Priestley gives a remarkable case, where the ovum never penetrated the decidua and thus developed outside of it. Of course a chronic metritis would affect the development of the membranes.

**Cystic Disease of the Chorion** is a very remarkable affection; it was formerly called hydatid disease of the uterus, for it was supposed that the cysts were hydatids, and grew in the uterus; indeed, no doubt it would often be difficult to ascertain their source, so that when, after a supposed pregnancy, the woman was about to be delivered, and instead of a fœtus hundreds of bladders or cysts were expelled, it was natural to suppose that these were either parasites or a multitude of human ova. But very often the whole came away in a mass, and then their connection with the membranes was seen, as in our many examples. We observe that they resemble large bunches of grapes, and are of various dimensions, though the cysts never reach any great size. If we examine them, we find they are formed by dilatations of the villi, not that there is merely one at the end of each villus, for we see a succession of the cysts, one above another, resembling a string of beads. This has suggested an origin by the budding of one from the other, a supposition which gave the title "proliferous cysts" to these bodies; but Dr. Braxton Hicks has contested this belief, and is of opinion that the production of them in rows arises through the cystic change coming upon many different points in the course of the long vascular tufts. Even where we see little buds starting from the cysts, like one cactus leaf from another, he thinks these buds did not grow after the cystic change, but had already formed when overtaken by it.

As to the pathological structure of these bodies the word cyst scarcely applies to them; for though very full of fluid, they have a solid meshwork. This consists of delicate fibrils with a few stellate cells, while the fluid which distends the "cyst" contains mucus, as was shown by Virchow, who considers that the mucous "Wharton's jelly" of the umbilical cord is continuous in the placenta with the substance of the villi. On these grounds he classes the "cysts" as myxomata, and, indeed, considers them as excellent types of the class. The same observer found in a full-grown placenta a large lobulated mass of a like myxomatous structure.

How the cystic change comes about is one of the most strongly contested questions of pathology. The usual view at present is that there is first a primary disease of the placenta, which itself is the cause of the death of the embryo or fœtus; Virchow suggests that it may arise from endometritis, or otherwise from some evil influence of the maternal blood or tissues on the ovum. On the other hand, Dr. Graily Hewitt supports the older view that the change is really a dropsy of the chorion villi, due to death of the embryo or fœtus, and perhaps showing itself long after that occurrence. In favour of this view it is true that the dead fœtus is very rarely to be found in such cases, and in considering their history they are often traceable to partial abortion about the eighth week, while similar changes may appear in portions of placenta retained after the full term. This seems the more probable view, and yet several circumstances cast doubt upon it; especially it is urged by Virchow that long retention of the placenta of a dead fœtus is not sufficient explanation of the change; for in extra-uterine pregnancy, and in other conditions where the placenta remains long in the body, it does not undergo these changes; indeed, the retained placenta is known to assume a compressed form; again, where the cystic change is supposed to have occurred in retained placentæ after childbirth, better observation has proved the cysts to belong to an abortive twin fœtus or a superfœtation; while, lastly, a partial cystic change has been found in the placenta at full term in the presence of the living fœtus. This is the most cogent fact of all; thus the question must at present be left uncertain. It is said that the affection implicates the whole surface of the chorion if it commence before the formation of the placenta; if afterwards, it is limited to the placental region. Virchow mentions a case where the placental attachment was the only part unaffected.

In St. Thomas's Museum there is a fœtus of four months surrounded by a cystic chorion, but the placenta must have contained many healthy villi so as to have nourished it.

The *amnion* may be diseased, but this has more to do with practical obstetrics; an excess of fluid is called dropsy of the amnion or "*hydramnion.*" It has nothing to do with dropsy either of the mother or fœtus, but it appears to depend on local causes. It may affect one of twins. Sometimes blood may be found within the amnion. More rarely an adhesion of it to the fœtus has been found. These adhesions are of great importance as causes of malformation. Cord-like bands may constrict and amputate the limbs, and local adhesions of the fœtus to the amniotic surface may cause, or at least go with, non-development of the affected part, as of one side of the head, &c.

**Extra-uterine Pregnancy.**—Sometimes, instead of the impregnated ovum arriving safely at the uterus, it is delayed in the Fallopian tube, where it undergoes development, either in the portion of the tube that is in the uterine wall, in which case it is called "*interstitial*," or else in the free part of the tube, when it is called "*tubal*," or it may be attached only to the fimbriæ of the tube; or it stays free in the abdominal cavity, and fails to enter the tube at all, when it is called *abdominal.* It is even said that the fœtation has happened in the ovarium itself, but we have never witnessed an example of it, unless, indeed, the hairs, teeth, &c., sometimes found in ovarian cysts, might be the result of delayed impregnated ova—an opinion that cannot be maintained, since similar growths are found in other parts. We have several times noticed an adhesion of the affected tube to the omentum, &c., as if it had been impeded in its function by this. We have one example of interstitial pregnancy, but of the tubular kind at least a dozen. We can see the embryo in all stages of development in the several cases; in some death of the patient has occurred as early as the sixth week, while in others the child appears almost full grown. In one curious case two fœtuses of different ages have been found attached to one tube. In other cases abdominal and uterine pregnancy have occurred together and reached their full term.

When the ovum is arrested in the tube all its parts become fully formed, as in the uterus; a good chorion surrounds it, which gathers nourishment from the tube, this having a large supply of vessels from the spermatic arteries. It has still to be determined in what way the connection is formed; it was the opinion of the late Dr. Robert Lee that a little whitish soft material, which we find enveloping the chorion, is in reality a decidua, and from this he argued that the deciduous membrane, in all cases of pregnancy, is a part of the fœtal structure rather than the uterine. In many cases there is not even this, and the villi simply lie stretched over the maternal vessels, which ramify on the surface of the Fallopian tube, the vessels or capillaries being immensely enlarged. In the interstitial case where the ovum lay in a cavity formed in the wall of the fundus of the uterus, the surface was smooth and covered with uterine vessels. There were therefore only the delicate walls of the blood-vessels between the maternal blood and the placental villi. It is remarkable, however, that these cases of extra-uterine fœtation have been taken as a corroborative proof that the decidua is formed from the surface of the uterus (if it be not, indeed, its altered mucous membrane), for it will be seen in all our examples that the uterus is lined by a thick, pulpy substance, which has a cribriform appearance, being pierced with a number of holes corresponding to the tubular glands of the organ. This is generally looked upon as the deciduous membrane developed in the uterus whilst the ovum is growing without; the uterus itself, we see, is enlarged, although containing nothing. It is even now a contested question to what extent the uterine mucous membrane is thrown off in the formation of the decidua and renewed in the course of pregnancy, some holding that it is quite exfoliated, as Cruveilhier thought. Others hold that there is a partial exfoliation about the third or fourth month, while in the later months the membrane grows again beneath, covering in the muscular tissue, so that this is not exposed at birth, but is lined with a thin, new mucous membrane; most good authorities agree with this opinion, we believe.

There are some who maintain that a deciduous membrane is formed every month, and that it comes away in disintegrated portions with the menstrual fluid. If a distinct membrane be found it is unnatural, and gives rise to dysmenorrhœa. There are others, however, who believe that menstruation is only a shedding of the epithelial surface, and that the deeper mucous membrane is untouched.

As regards tubal or *ectopic* pregnancy, Lawson Tait believes that when the ovum is retained and is developed in the tube it is because a previous salpingitis has occurred, and that a portion of the tube has been deprived of its ciliated epithelium.

It is a remarkable circumstance that in several of our specimens the corpus luteum is on the opposite side of the body to the ovum. This would suggest that the Fallopian tube must have sent its fimbriated extremity over to the other ovary in order to seize the ovum; of the possibility of this we are not certain. Or the ovum must have passed into the uterus, and then again made its exit on the opposite side; unless it be that the development of this body occurs indifferently in any Graafian follicle which discharges its ovum about the time of impregnation, whether it be the one actually implicated or not.

In most cases, when the ovum has reached any size, and the chorion has begun to collect into one mass to constitute the placenta, the tube bursts, and death takes place very rapidly by hæmorrhage; if, however, only a slight lesion should occur, an inflammatory process may take place, causing an adhesion of the containing sac to surrounding parts, while development goes on. When the full period of pregnancy is reached, and the fœtus is unable to escape (the same occurs if it should die prematurely), it acts as a foreign body; adhesions are formed to surrounding parts, suppuration occurs, and the bones and other portions escape by openings in the abdominal walls or by the rectum; in one case, at the end of gestation, a woman was taken with the usual signs of labour, which passed off in a week; her health then began to fail, abdominal pains distressing her; after three years an abscess broke into the rectum, and some fœtal bones escaped; these continued to come away for another three years, when the woman recovered. Sometimes, however, the fœtus has dried up or mummified, and remained quiet for several years; we have a specimen showing a mass of bones which were long retained in the abdomen; but sometimes the fœtus is changed into adipocere or infiltrated with calcareous matter. A specimen is to be found in the College of Surgeons, and has been called a stone fœtus.

## PLACENTA

As the diseases of the ovum, and especially of the chorion, have to do with abortion, so do the diseases of the placenta constitute, very frequently, the causes of miscarriage. Sometimes the changes in the placenta appear to be merely a premature condition of what is natural at the end of the ninth month of pregnancy; at this period the placenta separates from the uterus like fruit from a tree, having lived its natural period of life, so that it is suffering the same process of decay which the whole body does in old age; thus an earthy matter is constantly formed in it, and the *placental tufts undergo a fatty degeneration.* This has been disputed, yet the occurrence of some degree of fatty degeneration here is so constant as to leave no reasonable doubt that it is natural at full term. But if this change should occur prematurely Barnes has shown that a miscarriage is the result, and then, if portions of the placenta be put under the microscope, the degenerative changes may be discovered, or the morbid deposit may even be seen as white specks by the naked eye. Generally, however, during the gradual separation of the fœtal and maternal structures, some oozing of blood takes place, and then, besides much that escapes externally, the placenta may be found occupied by large coagula—a condition to which the name "apoplexy of the placenta" has been given. As, however, the separation is gradual, the blood has time to undergo various changes, the colouring matter is taken up, and the fibrin is left. It may be, however, as Virchow urges, that such fibrinous masses are more frequently the result of coagulation of blood in the maternal part of the placenta than of actual apoplexy. In many of our specimens the placenta, instead of being spongy and soft, is hard, and of a yellow-white colour; the structure within is firm, and the vessels and villi are with difficulty separable, being united into one compact mass. This state is, however, present only in parts, and thus several distinctly circumscribed lumps or tumours are formed within it. If these be carefully examined by the microscope after tearing up a portion of the tissue, the villi will be found charged with granular and fatty matter, and the fibrillated and granular material of old blood-clot will also be seen between them. We may notice in the Museum that many of these diseased placentas were called tuberculous, for the reason that all unorganisable or amorphous deposit formerly received this name; but in most of them the pale solid matter is merely altered blood. In some cases, where the yellow masses have softened and broken up, the question of tuberculous disease has arisen. Sir J. Simpson gave the weight of his opinion to this view, and called the disease *placental phthisis.* Some of the hard, yellow masses met with are probably *syphilitic,* being composed of cell- and fibre- tissue, and resembling closely gummata found elsewhere; there has also been great thickening of the blood-vessels. Mr. Wilkinson King described, many years ago, what he believed to be syphilitic disease of the placenta.* Cruveilhier described, many years ago, an atrophy of the placenta in parts which led to abortion. It seemed to him a withering of the tissue from insufficient blood-supply. Owing to the resemblance of this adventitious matter to lymph, it is thought that the consolidation of the placenta corresponds with the similar condition of inflamed lung, and thus the term *hepatization* is given to it. In most cases, however, there is no proof whatever that the lymph-like matter is an inflammatory product; and yet inflammation of the placenta, or *placentitis,* does occur, and by it an exudation of lymph takes place which binds the placenta to the uterus. It is certainly true that a new cell and connective tissue may be found; some have even described suppuration. It is possible, however, that a liquefied thrombosis may have been mistaken for an abscess. This is the cause of those rare cases of adherent placenta which some attribute to falls or injuries during pregnancy. It is also very common to find calcareous degeneration of the placenta, even to a considerable extent; this is probably a truly morbid condition, and yet lower degrees of it at least are compatible with apparently sound health of the fœtus.

Other morbid conditions of the placenta, referring more to obstetrics, will be pointed out in the midwifery lectures; such as the effusion of blood between it and the uterus; also its malposition in the uterus, as placenta prævia, &c. Virchow has described a morbid condition, which he calls "myxoma fibrosum," where the villi become converted into fibrous tumours the size of nuts. Galabin thinks that some of the lumps found in the placenta are occasionally new growths, sarcomatous or adenomatous.

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* His cases are referred to in a paper by Dr. Wilks on "Visceral Syphilis" in the 'Guy's Hospital Reports' for 1863.

## UMBILICAL CORD

The various affections of the umbilical cord will also be explained; such as disease of the blood-vessels, or their plugging, leading to suppuration of veins and death by peritonitis, or occasionally by pyæmia with scattered abscesses on the body; œdema of the cord, knots in it, &c.

## FŒTUS

The diseases of the fœtus have of late years been regarded with great interest, not only in reference to any constitutional cachexia to which they may give rise, but as originating those early organic changes which we are in the habit of calling malformations; and when we speak of disease we do not use it in that general sense signifying a mere deviation from the healthy standard, but, in most cases, as a positive malady, such as occurs in extra-uterine life; as, for example, a local inflammation, or a rheumatism. Many malformations, however, must still be ascribed to aberrations in the developmental power, and this is especially true of such malformations as occur in the mesian line from an imperfect fusion of the two sides of the body, such as are seen in cleft palate, extroversion of the bladder, &c., as well as others which arise from excess of development.

These malformations we have already sufficiently spoken of in their respective sections, and we shall now just allude to some other abnormalities special to the fœtus which did not come under the descriptions already given.

A very remarkable set of malformations are those ascribed to spontaneous amputations *in utero*, supposed to be caused by the umbilical cord becoming twisted as a ligature around them. The effect of such twisting could hardly be a complete severance of the limb, for such tight constriction would probably stop the circulation in the cord and so lead to the death of the fœtus. But it has been shown to produce grooves on the limb affected, and may, by stopping the supply of blood, prevent the growth of the distal portion, so as to reduce it to the dimensions of a mere bud on the end of the proximal part. This is probably the explanation of the bud-like growths from the ends of amputated stumps which have been thought to be attempts at reproduction of the lost part.

The actual severance of the limbs has been explained by Dr. Montgomery as due to constriction by bands within the amnion; numerous observations have confirmed his view. The separated part, however, is generally not found, but the amniotic liquid is then turbid in some cases, suggesting that it may have disintegrated. A more probable cause of amputation is constriction by the cicatrix of a cutaneous ulcer. Such ulcers have been occasionally met with in the fœtal integuments.

Intra-uterine fracture by violence is another cause that has been thought capable of causing amputation, but it appears very improbable that the yielding tissues of the fœtus would be broken by an injury that did not produce abortion. Other cases are unexplained, and some think then that amniotic strings which did the mischief may have disappeared. In cases of amputation we sometimes see signs of a scar on the end of the stump. But there are a series of cases where the limbs are cut short that do not admit of explanation by supposed amputation—cases, that is, where the deficiencies are symmetrical and affect many joints. In the case from which some models were taken, in which the hands had lost many fingers, the losses corresponding on the two sides, the man said the malformation was hereditary in his family for many generations; he said, too, that the deficiency always happened in the short members of his family, while those with perfect limbs were always tall, some of them being in the Life Guards. Partial development of a limb, however, must be accounted for on other principles, especially when the disorder follows some anatomical district, as, for example, when the fibula and toes on the outer side of the foot are deficient; withering of a limb may arise from paralysis; and in such a case we have seen an atrophy of the opposite side of the brain as the cause; in a similar case which we have already mentioned, where death occurred during birth, an effusion of blood had taken place in the brain, and it was directly traceable to a fall the mother had suffered during pregnancy.

With respect to the *heart*, the various malformations, though so different in kind, can be for the most part well understood and classified on the supposition of some endocarditis during fœtal life; the proof, however, that the fœtus had rheumatic attacks is still wanting, although in one instance of malformed heart there is a history of the mother having rheumatism during gestation.

We have already spoken of *brainless* infants, and cases where from a hole in the skull a part of the brain or the membranes escape, constituting *encephalocele* and *hydrencephalocele;* also of *spina bifida*, and its association with club-foot. But besides such malformations, the brain and spinal cord in the fœtus are often found *diseased.* Virchow describes a *congenital encephalitis;* the disorder has the character of white softening—that is, we find in it granule-cells; while the tubular tissue is broken down, its fibrils being sometimes separated into short, rice-grain-shaped bodies, which we have frequently seen. The evidence of its inflammatory nature is not more conclusive than in the white softening of the aged; but Virchow maintains that view on the ground that such white change coexists with inflammatory processes in the kidney and elsewhere; also that it is often caused by scarlatina, smallpox, and syphilis. Some degree of this condition is common in syphilitic infants, and it may be found at any time during the first few months of life; its signification in explaining malformations and imperfections in the development of the limbs is evident enough.

In coming next to the *skin*, we find here specimens which we have not yet alluded to, and amongst them are our three remarkable examples which go by the name of *harlequin fœtus*, or skin-bound fœtus, or *intra-uterine ichthyosis.* They are not very common, so that our Museum may be considered rich in such specimens. They are all at full term; the skin is covered with fissures, which, crossing in all directions, give the resemblance to a harlequin. The impression which is first conveyed to one's mind by looking at them is that the skin had ceased to grow at a certain period, while the tissues within, continuing to increase, had caused distension even to bursting, and thus the integument is cracked and fissured on the most prominent parts of the body. As we do not know that they have ever been described, we will point out more in detail some of the peculiarities which are seen in our specimens. The fœtus is of moderate size, fully developed, and the skin covered with fissures; these are more numerous in front than behind; the lines have a general tendency to be horizontal around the body and abdomen; on the chest they run in oblique directions, producing lozenge-shaped spaces; on the extremities their course is transversely around the limbs; in those parts where the body is flexed, the fissures are just in the direction which would follow from the stretching. The face is fissured in all directions; on the head the cracks pass from back to front; on each side of the body the transverse lines are intersected by wide longitudinal ones. All the fissures have an appearance of having been caused by stretching; in some parts merely the cuticle is affected, but in the deeper fissures the true skin is torn, and we see fibres passing from side to side, just as would be produced by a forcible separation of the skin. In one or two places it appears as if the cuticle had been destroyed and again repaired. The fissures are about one eighth of an inch wide. The eyes are perfect, but the eyelids fixed back to the forehead, so that the conjunctival surface is exposed; there are no eyelashes; the nose is not much altered, the mouth is kept open by rigidity of the skin around, which is as hard as cartilage, and forms a complete rim to it; there is no external ear; there is hair on the head; the fingers and toes are perfect, but are drawn together by contraction of the skin.

Mr. Bland Sutton suggests that at the fourth or fifth month of intra-uterine life there is an excessive activity of the sebaceous glands, so that an undue quantity of "vernix caseosa" is formed; that this cakes on the skin, hardens, and deforms it. With this view he would call the disease general seborrhœa.

There are also other cutaneous affections which may occur in the fœtus; for example, we constantly meet with cases of syphilis where the cuticle peels off; and we possess a fœtus which was the subject of smallpox. We just now mentioned the probable effects of cutaneous ulcers in causing spontaneous amputation *in utero* by their cicatricial contraction.

As regards the *lungs*, we have seen in many instances acute lobular pneumonia in the fœtus at from six months' development to full term. Such pneumonia may be found in the form of lobular grey or white hepatization. It is most frequent in syphilis; white lobular hepatization is thought by some to be characteristic of syphilis in infants. We may remark that we have never seen tubercle, and we believe that congenital tubercle has not been met with.

In the *peritoneum* many important changes occur from fœtal inflammation, and that this is not uncommon was shown many years ago by Sir J. Simpson, who found it in children born of syphilitic mothers; it might have been thought that the wretched puny condition of such fœtuses was quite sufficient to show their incapacity for life, but in truth there is very often an acute internal inflammation which has probably been the immediate cause of death; thus we have now examined many syphilitic infants born dead, and in most of these there was evidence of peritonitis; in two, old adhesions between the liver and the diaphragm, and in the others a general inflammation; in two there was also lobular pneumonia. In the adult we may constantly meet with adhesions, when there has been no history of peritonitis since birth, so that they probably have their origin in the fœtal state. It has also been suggested by Sir J. Simpson whether the malpositions of the intestine are not due to local peritonitis; and there can be little doubt that such is the case, for wherever these are met with, adhesions are always found binding the errant part in its new position; we allude especially to those cases of which we have lately seen three instances, where the ascending colon and cæcum were dragged over to the left side of the body; in one case there was a discussion as to whether this was due to a malformation or to an inflammatory process; but if we consider the altered position to be due to an adhesion in fœtal life, both suppositions are true. It has also been thought that the non-descent of the testis may have been due in some cases to peritoneal adhesion, especially where a coil of intestine has been found connected with the internal abdominal ring. Sometimes the intestine is open at the umbilicus, as in a case related in our 'Reports;' this is occasioned by the umbilical vesicle, which in fœtal life is connected with the intestine, not closing as it should do.

The liver is naturally of large size and dark colour in new-born children; it is but little subject to disease in the fœtus, except in congenital syphilis; in this condition it has been found to show gummatous patches. This we have ourselves never seen, but there is another more frequent form of congenital syphilitic hepatitis in which the organ is pale and hard, being elastic and bloodless. The disease is due to the production of new inflammatory matter in the form of organisable round and spindle-celled lymph in the tissue of the liver about the portal veins. By the pressure of this the proper tissue perishes, but very evenly, so that the surface is smooth and the section uniform. The organ becomes remarkably hard and solid, but the affection is not very conspicuous, and may be overlooked unless great care is exercised.

The kidneys are lobulated on the surface. We have spoken of the congenital cystic change occasionally found in them. It is not uncommon to find concretions of uric acid and urates in the pelvis and on the pyramids of the kidney.

**Monstrosities.**—*Teratomata.*—These may be of various kinds; sometimes mere masses of organic matter, with only a slight resemblance to a human being, may be looked upon simply as imperfect growths. The most remarkable monsters, however, are those where a body exists with some extra portions attached, and very diversified opinions are held as to whether these be due to an excessive germ power or to a union of two ova, of which one only is perfectly developed. In the case, for example, of the Siamese twins, there can be no doubt that two fœtuses are simply united; while in instances of six fingers to a hand the case cannot be looked upon otherwise than as an excessive development of one fœtus; but if an extra limb be found protruding from the body, a difficulty arises as to whether one or two germs were involved. The union of two fœtuses is clear in cases where the bodies are separate but the heads united. It is evident too that one ovum may be included in another, and thus we might have the case, as in the parasite lately exhibited at the Pathological Society, where a well-developed man has the four limbs of another person protruding from his body. It only requires the limbs of such a case to be joined together to produce the three-legged monster which was made a public exhibition of some years ago; the middle limb had ten toes, and the child, which was alive, had double genital organs. A smaller limb is here represented; it was removed by Mr. Cock about two years ago; it grew, as seen from the lower part of the abdomen, and when cut contained a long bone, like the tibia, running through it; the fact that these occur in the median line of the body suggests that they belong to a second ovum. Sometimes this second ovum does not project in the form of additional limbs, &c., but forms a tumour more or less included in the body; if so it is generally a mere shapeless mass, or has little pretensions to the figure of a human being; as seen in our model of the curious fœtus found attached to the stomach of a boy, and also in drawings of several other recorded cases. We have in a jar a quantitiy of bones which were taken from the buttock of a child, and which must have belonged to a twin fœtus. When we come to smaller protuberances from the body, the question arises, as we have before said, as to their being portions of a second ovum, or due to excessive power in the single ovum; as, for example, in a curious body, removed from the back of an infant, which was called a tail.

A very favourite place next to the ovary for embryonic or fœtal growths is the sacrum, and many cases are recorded of the curious composition of these tumours. Dr. Pye-Smith had a case of a child in whose abdomen was a cystic growth containing fat, hair, bone, and rudimentary feet. On commenting on the case he was disposed to think that these were the products of a second impregnated ovum included at an early period of development within the twin embryo,—a second impregnated ovum attached to the first, and therefore of the same nature as the Siamese twins or the two-headed nightingale.

Mr. Bland Sutton has shown that a large number of tumours and new growths have their origin in embryonic structures, as, for example, in connection with the pituitary body, branchial arches, Wolffian bodies, &c., and therefore some of these congenital tumours found between the rectum and coccyx have originated in some embryonic structures connected with the development of the intestine.

# TUMOURS

The word tumour signifies simply a swelling; and there, perhaps, was a time when that was all that was meant by it, but every swelling is not now called a tumour. Thus, the common unsightly swelling of the face, from carious tooth and abscess, is never called a tumour. Yet the exactly similar appearance caused by growth from the gums or facial bones is a tumour. Again, ascites is never known as a tumour, and that, not because it is better known as dropsy, for the limited dropsy of hydrocele is called a tumour. So that swelling is not what is meant by tumour; indeed, some of the most dreaded tumours often shrink the part they attack; as, for instance, scirrhus of the breast and certain puckering cancers of the peritoneum.

A strict definition of the word tumour is, perhaps, impossible. In fact, a pathologist trying to define a tumour is like a botanist trying to define a shrub; for just as the word shrub was never meant to be botanical or scientific, being simply "what would grow in a shrubbery," so "a tumour" was never meant to be a pathological or scientific term; it means an obstinate, persistent, unmanageable swelling, that must be removed if possible.

The kinds of swelling which it is agreed to call tumours have the following set of characters:

1. They arise by a new centre of growth.
2. They organise in solid continuity as they grow.
3. They persist.
4. They form circumscribed, or, at least, easily determined swellings.
5. They are abnormal, so that the tissues of which they consist, although often resembling them, are arranged differently from the normal ones, and have not their function; as, for example, as is seen in the adenoid tumours.

Now, this is, we think, the best way of solving the question, "What is a tumour?" The *beau idéal* tumour would include all these characters. Such a tumour is an adenoma of the breast, or sarcoma of bone. No one would hesitate to give the full force of the word tumour to such a growth as possessed all these characters.

But many things that lack one or more of these characters are still called tumours. Thus:

1. Some tumours have no new centres of growth, but are enlargements of existing parts, such as splenic and glandular tumours.
2. Some tumours do not undergo organisation, such as ranula, wens, and hydrocele.

3. Some shrink the part they grow in, such as scirrhus and peritoneal cancer.

So that many of the things called tumours corespond but partially to the standard of the most perfect tumours, which develop from centres, and organise themselves like new organs in the frame; and thus they lose many of the characters of those perfect tumours, until such structures as syphilitic gummata and large tubercles are reached, which we should scarcely call tumours, and yet hesitate to deny the name.

As to the cause of the origin of tumours, the question rather belongs to theoretical pathology than to morbid anatomy.

There has been thought by some to be a state of mixture of the blood, such that the substance of the tumour comes out as a *residue*, when the other organs have taken their respective shares of nourishment. By others that there is an organising power in the *blood*, so that it actually pours out material that forms a tumour. Then there is the belief that there is a growing power in the solid elements of *texture*, which creates the tumour by augmenting such elements; and it is also held by some that there is a kind of *parasitic* nature in the life of the new growth.

Then, again, there have been various theories in explanation of the special characters which any tumour or new growth assumes, which growth we might say has received the more scientific appellation of persistent or growing neoplasm, as distinct from a hyperplasia which is generally the result of inflammation, and is attended by symptoms during its formation. There are some pathologists who would refer the character of new growths to the kind of elementary tissue whence all the structures of the body spring. With this view the epiblastic, mesoblastic, and hypoblastic, embryonic layers would each have their own special series of growths which would necessarily vary in the different structures of the body according to the layer from which these arose. We see then most tumours consisting of the simplest structures, such as cells, fibre, or cartilage, or having their origin in the ancient or primary elements of the organism. If, therefore, it be held by some that cancer is nothing more than a mass of embryonic cells which might have developed into higher tissues under ordinary circumstances, it is not remarkable that the term malignant should be given to it, since we observe nothing but a mass of cells growing and extending in all directions in a purposeless manner, destroying and eating away everything as it proceeds. Cohnheim went further, and suggested not only that tumours spring from tissue according to their original embryonic nature but from distinct portions of embryonic tissue which had remained in an undeveloped state, that portions or traces of this might be nascent in the system until some special deteriorating influence would bring them into activity, when they would start into life. We have already spoken of growths in the ovary and testes which might have such an origin, as well as some congenital tumours of the kidney. This doctrine is not regarded by most as proven, whereas some of his more general opinions are accepted, as that new growths always spring from certain tissues, as, for example, the carcinomatous from the epithelial, whilst the sarcomatous have their origin in the connective tissue, and so on. It may be said generally, that the more the structure of a tumour is allied to a natural one the more localised and innocent it is, and the nearer it approaches to a simple cell formation the more malignant it is in its nature, returning when removed or infecting the system. The benignant was at one time called homologous and the malignant heterologous, but as these terms imply the existence of different kinds of elements, the more modern words, "typical and atypical," have been used as denoting that the tissues are arranged or not arranged according to the manner of normal structures. A tumour recurring after removal, and spreading through the body, has the name "malignant" given to it, to imply that its formation is due to some vice in the whole system, and in corroboration of this view it has been surmised that there may be a special carcinomatous bacillus or micro-organism which starts it into life. On the other hand, it is clear that local influences are powerful in determining the nature of a new growth; as, for example, when a tumour containing bone is seen to have its origin in the bony skeleton, a myeloid in the marrow, and a melanotic tumour in a pigmental structure. Epithelial cancer, too, in a most striking manner displays its local source, for although sometimes it is found in the liver, lungs, kidney, and other organs, it invariably has its origin in epithelial structures. The local influences, therefore, which are at work in determining the character of the new growth are evident at the same time that the general infection of the system is seen to be equally manifest. The belief of some that there is in all probability a microbe which causes the propagation of cancer does not seem a necessity with others, seeing that a cell with its contained elements is as vital and capable of growing as a parasite. And even if no constituent of the cell is carried as a seed to spring up elsewhere, it might carry some influence as the sperm does to the germ, and so cause a healthy cell to put on a new character. In this way only can one understand the view taught by Creighton and others that a healthy hepatic cell may become a cancerous one. The reconciliation of two apparently opposite facts is a desideratum of great moment, but with the words of Sir J. Paget we must at present be content. "I think malignant tumours are local manifestations of some specific morbid states of the blood, and that in these are incorporated peculiar morbid materials which accumulate in the blood and which their growth may tend to increase."

Whatever theories of the nature and causation of tumours we may hold, it is found most convenient to classify them according to their structure; that is, on an anatomical or histological basis. But it will be seen how rarely any of the structures occur in their simplicity, as, for example, in the case of osseous tumours which may contain cartilage, bone, fibre, and blood-vessel all combined.

They may be divided into :

1. Cysts.
2. Osteoma ; osteoid chondroma ; enchondroma.
3. Fibroma, sarcoma, glioma, myoma, psammoma.
4. Myxoma, lipoma, myxochondroma.
5. Adenoma.
6. Carcinoma.
7. Lymphoma.
8. Angioma and lymphangioma.
9. Teratomata.

**Cysts.**—Some cysts own a very simple origin ; they are mere enlargements of natural cavities or channels. Thus, natural cavities may be distended with fluid ; for instance, that of the tunica vaginalis, which then forms the cyst called hydrocele. Such cysts are distinguished by Virchow as *exudation cysts.*

The channels of ducts becoming obstructed, a dilatation of the duct or of the follicles of the gland behind the impediment occurs, and then are formed *retention cysts.* Numerous instances of these, such as wens, ranula, &c., we have already alluded to. Those of the follicles are seen in the mucous membrane of the stomach, œsophagus, lips, tongue, vagina, &c., and are called *mucous cysts.*

Hæmorrhage also gives rise to cysts, as in the external ear in the so-called hæmatoma auris (see " Ear "), in the brain, in the substance of tumours, &c. These have been called *extravasation cysts.*

Other cysts arise in tumours by the softening down of the texture ; these are not infrequent in enchondroma, myxoma, fibroma, sarcoma, adenoma, and carcinoma. The resulting cavity contains at first a turbid, often mucoid fluid, but this becomes clearer afterwards and the walls of the cavity may become quite smooth—*degeneration cysts.*

Besides such exudation, retention, extravasation, and degeneration cysts, whose origin is capable of being demonstrated, there are other cysts which appear to arise spontaneously as new growths.

Some others appear as enlargements of the remains of fœtal organs, such, for instance, as encysted hydrocele of the testis, cysts of the broad ligament, &c., and *dermoid cysts.*

Others are overgrowths of the natural elements in a tissue, such as the Graafian follicles, and, according to some views, a variety of renal cysts.

Others yet appear due to distension of the areolar tissue spaces, as encysted hydrocele of the cord, cystic myoma of the uterus.

Again, dilatations of the lymphatic channels form tortuous spaces in the hypertrophied subcutaneous tissue, as of the thigh occasionally.

Other cysts are formed as in the liver, brain, &c., by simple vacuolation.

**Osteoma, Osteoid Chondroma, Enchondroma.**—These compose a sufficiently natural group of kinds; indeed, it is often practically difficult to refer a tumour to one of these kinds, they are so apt to be mixed together in the same tumour. However, when bone forms a large part or apparently the whole of a tumour, the tumour is called an *osteoma*, but no tumour is ever really formed altogether of bone; there is always present an ossifying matrix, by the ossification of which the bony part of the growth enlarges. So that the growth is not of bone, but of the soft part which precedes it, and which changes to bone when it has already grown, and only so far as it is not any longer growing. The kind of matrix varies much, and the structure of the whole growth takes its vital properties from the matrix. Thus, sarcomata or even carcinomata may directly ossify, and so we may get *osteo-sarcoma* and *osteo-carcinoma.* But when this is so, the bone is in relatively small amount, and of still less comparative importance; the tumours are practically sarcoma and carcinoma, and the kind of interest that attaches to the bone in them lies rather in this, that such tumours arise almost always from bone, and we see in the bony development an evidence of the continuance of the nature of the mother tissue into the nature of the offspringing tumour.

The forms of matrix which produce growths of practically a bony nature are generally two, viz. fibre or periosteum, and cartilage. Periosteum—or, to speak more exactly, a tissue resembling closely the deeper layer of the periosteum—forms large tumours, whose transformation into bone takes place by the cells putting on the shape of bone-cells, and, the matrix calcifying, these tumours are called *osteo-fibroma*, *osteoid chondroma*, or *periosteoma.*

Cartilage forms large tumours, generally arising from bones, especially those of the extremities, and more especially of the distal parts of the extremities; these grow generally within the bone, more rarely under the periosteum, and form large rounded or lobulated tumours. To the unaided senses these tumours offer plainly the well-known characters of cartilage, except that they are nearly always divided by septa into lobules; the consistence varies, and when large they may be in parts very soft and sticky, or mucous, or softened cysts; when this is so, their structure varies towards that of mucous tissue, whence they are called *myxo-chondroma*, but, instead of softening, a large enchondroma often hardens, and appears to be ossified, though it is only petrified by deposit of calcareous salts in its matrix; this change is, as is well known, the first step in ossification of cartilage. In many cartilage tumours the process goes no further, or it may proceed through the several stages of vacuolations, formation of medulla-cells in the vacuoles, and direct transformation of these to bone-cells. More rarely the cartilage-cells, without calcifying, proliferate and change directly into bone.

The amount of cartilage, periosteum, or bone present varies indefinitely in different cases. When cartilage preponderates, the tumour is called an *enchondroma ;* when bone preponderates, an *exostosis*, *osteoma*, &c., according to its shape and connections; when periosteum preponderates, an *osteoid chondroma*; if the ossified tissue is simply fibrous, it is called *osteo-fibroma.*

Occasionally in tumours that project from the surface of the bone, as of the femur, &c., the amount of bone and cartilage is so equal that it is a matter of difficulty to decide which name shall be used, and then the terms *cartilaginous exostosis*, or *ossifying enchondroma*, are employed. *Osteoid chondroma* is to be suspected of malignancy ; it makes a part of what were called *osteoid cancers*, being liable to set up similar growths in remote parts, as the lungs, &c. All these different tumours have already been considered under section "Bone."

**Fibroma, Sarcoma, Glioma, Myoma, Psammoma.**—These form a natural family inasmuch as they correspond in structure to the fibrous and connective tissues, including those forms of connective which enter into the composition of the organs, and particularly the neuroglia or connective substance which unites the proper nervous elements in the brain.

The distinctive character of all these kinds of tumour is the possession of a structure microscopically homogeneous, and consisting of cells with an intercellular matter surrounding them, so as to be between them all. The qualities of this intermediate tissue between the cells determines the kind of tumour. Thus, in *fibroma* it is fibrous, in *sarcoma* it is soft and slightly fibrillated, in *glioma* it is yet softer and more delicate, being sometimes granular and almost fluid, at other times showing a felt-work of extremely slender long fibres. The proportion of cells varies extremely ; sometimes there are few, as in some fibromata and gliomata, which are almost wholly fibrous.

The larger the cells and the greater the proportion of the whole tissue they compose, the more is the sarcomatous character developed. To understand the relation of sarcoma to the other kinds, we must remember that in all the connective class of tissues the character of the tissue depends on the intercellular matter. This is strongly fibrous in tendons, more delicately fibrous in areolar tissue, imperfectly fibrillated or quite homogeneous in the papillary layer of the corium, extremely soft in the brain, &c. The cells within it in these normal tissues are so few as commonly to escape notice, but in the formation of tumours corresponding to either of these kinds the cellular elements increase disproportionately, while the intercellular substance is not only thus relatively less in amount, but also is deficient in character.

Now, when the increase of cells and alteration of the intercellular substance are not so great but they still leave the main characters of the tissue recognisable, the tumour is called after the tissue it thus resembles, a fibroma, or glioma, &c. But when one of these tissues is produced very rapidly it has no time for its intercellular matter to acquire the proper characters of its normal structure, and hence it remains intermediate, while it also is small in quantity, the cells greatly preponderating. This constitutes sarcoma; indeed, we should find that any of the previous group of tumours would become sarcoma by rapid growth of the cellular elements and deficient development of the intercellular matters, and thus osteoma becomes osteo-sarcoma, &c.

In this way any of the normal connective tissues may produce by rapid development a tumour of sarcous tissue or sarcoma (the name is well chosen, σαρξ, which equals *caro*, or our word *flesh*, means commonly any soft animal substance, not blood nor bone). If follows that while yet the character of the original tissue is recognisable in the tumour, this will qualify it, so that there will be several kinds of tumour approaching sarcoma according to the tissue of which each is a development, while the condition of pure sarcoma is a culmination of the rapidity of development, such that all character of the intercellular substance is lost.

These tumours, as we have said, are named according to the tissue they are derived from or resemble. Thus, those from lymph-gland are called lympho-sarcoma, those from the neuroglia glio-sarcoma, those from the fibrous and areolar tissue fibro-sarcoma, and those from bone osteo-sarcoma. In either case, when the character of the original matrix is lost, they are merely called sarcoma.

A simple sarcoma is composed of embryonic connective tissue in which is contained an intercellular substance. It is thus a connective-tissue growth just as cancer is one springing from the epithelium. The simple sarcomas are divided into the *round-celled* and *spindle-celled* sarcomas, and the former again into the *small-celled* and *large-celled* sarcomas. The small-celled are soft and the cells are the size of the white cells of the blood. The larger-celled are firmer and have large nuclei.

It must be remarked, in microscopic observations of various sarcomatous tumours, that cross-cut bundles of spindle-cells appear as groups of round-cells, so that it requires careful focussing to distinguish one from the other.

Tumours of this group appear to the unaided senses either as evidently resembling the tissue from which they are named, or more or less soft and fleshy in proportion as they approach the condition of pure sarcoma. As they deviate from their normal type they tend to be subpellucid and elastic. They yield no milky juice when the section is scraped, as we shall find that carcinomata do.

*Glioma*, or tumour of the neuroglia, is almost peculiar to the brain and nerves, in which association we have already described it.

*Psammoma*, or sandy tumour, is a name given to certain rare growths, in which calcareous salts are found in the fibrous basis of the tumour, making it gritty or even sandy in section. They are almost limited to the membranes of the brain and spinal cord.

*Myoma* resembles sarcoma, but the cells develop into non-striated muscular fibre-cells. These tumours are almost strictly limited to organs of which non-striated muscular fibre forms a considerable part; they are especially frequent in the uterus and prostate, but may be found throughout the alimentary canal occasionally. We have already mentioned the congenital tumours of the kidney which contain striated muscular fibre, and Mr. Butlin exhibited at the Pathological Society a fatty tumour from a child which contained muscular fibre. This was probably congenital, as probably are all which contain true muscle. They are therefore practically of two kinds: the non-striated, found mostly in the uterus, and the striated, of an embryonic nature.

*Melano-sarcoma* is a name applied to sarcomatous tumours which are coloured brown or blackened by a deposit of pigment in the cells. Such black tumours generally arise in parts where pigment is naturally present, as in the eye, the pia mater, and the skin. Except for the blackness, the tumours have the ordinary characters and tendencies of sarcoma.

**Myxoma.**—The name myxoma is given to all tumours of connective-tissue type which contain mucus or mucin in their intercellular matter. It corresponds nearly to gelatinous sarcoma, collonema, and fibro-cellular and some colloid tumours of older authors. The forms of the cells are very variable, but in the most typical examples of such tumours, and especially in their older and fully developed parts, the cells are large and usually multipolar or "stellate," with a distinct nucleus and nucleolus; the stellate branching rays of the cells are mutually connected, so as to form a more or less open network, in the interstices of which the mucous semi-fluid lodges. Beams and bands, which generally have a stiff rigid appearance and an angular rather than a wavy disposition, pass about, dividing up the substance of the tumour into very imperfectly defined sections, more or less visible to the naked eye; from these beams arise fine fibrils continuous with the cellulo-fibrillar network. Cells can be seen embedded in the beams themselves, which may preponderate so as to give a fibrous character to the whole, acquiring the name *myxo-fibroma.* In any myxoma much of the tumour, and especially the younger part, may be found formed of spindle-cells. These are really connected, by means of threads from their sides, the threads joining together, and thus all that is required in order to produce the stellar figures of the typical and complete myxoma-cells is the drawing out of these threads to greater lengths, through the separation of the texture elements by an increasing quantity of mucus infiltrated into the tissue, and thus the compact spindle-cell parts are young, and the stellate parts middle aged. In yet other parts the prevailing form of the cell is round, or with one pole; and these round-cells resemble ordinary mucous corpuscles, like these being scattered in the mucoid matter, while the stronger fibrils still remain; such cells often contain many fat-grains, and are found in the oldest parts of the tumour, representing the senescence of the growth by fatty decay of its cells.

Thus, there are different degrees of softness in the several portions of a large myxoma, according to the various ages of its parts; the younger portions being more compact, and the older showing a greater quantity of mucus, until some parts may appear clear and pellucid, when the term *hyaline myxoma* is applied; or such soft parts may be turbid through the fatty cells present, while they are still gelatinous and tremulous in consistence. The softness of the fibres and cells may go on until a cyst containing mucoid fluid is formed; such a tumour would be called *cystic myxoma.* Although we so far describe the variation of consistence as developmental, and representing stages in the life of the tumour, some growths, undoubtedly, show a greater tendency to the breaking down of the solid web of cells and fibres, while others have naturally a much greater proportion of solid part in the form of strong bundles of fibres, which may make a close tough tumour, only known to be myxomatous by its stickiness; this would be called myxo-fibroma.

Other variations in the nature of the tumour may take it towards the characters of cartilage, thus producing *myxo-chondroma*, the cells becoming encapsulated and the interstitial substance chondrinous; or the cells may enlarge and fill with fat until the appearance approaches, or is identical with, that of adipose tissue, so forming *myxo-lipoma.* In other examples we may meet with large polynucleated cells, like those called giant-cells, which more specially characterise the kind of sarcoma once called myeloid; these are, however, rare.

Myxoma generally forms large soft tumours of elastic consistence; they are more gelatinous and tremulous in their consistence than sarcoma; the section is sticky, and its appearance generally more pellucid than that of sarcoma.

In the theory which supposes each tumour to have a type in some normal structure, myxoma is affiliated to certain natural tissues, in particular the jelly of the umbilical cord, the vitreous of the eye at a stage of its development, and the early stages of adipose tissue, as well as a stage of bone formation out of cartilage. It will be seen that these typical tissues are only transitory in their nature, as compared with such stable tissues as bone, cartilage, tendon. It is in accordance with this instability of their type that myxomas themselves should show transitions to permanent kinds of connective tissue, such as fibrous tissue, cartilage, or fat, as we have already mentioned. Tumours are not unfrequent, especially in the parotid region, which are thus intermediate between cartilage and mucous tissue, so that one cannot say to which they most properly belong; also many fatty tumours show clear gelatinous patches of mucous tissue in all transitions to fat, while some myxomata show opaque spots composed of true adipose tissue.

*Lipomatous tumours* are very common, either in circumscribed and encysted form, or diffused. They are not only found in the areolar tissue of the skin, but as already mentioned, in the mucous membranes of stomach, intestine, and other parts.

**Adenoma.**—The essential character of adenomata lies in the possession of a glandular structure; but the comparative amount of the glandular element varies much. They may be divided, like the natural gland, into the racemose and tubular varieties. The former is best seen in the breast, the latter is more common in the mucous membranes. They constitute a general hypertrophy, or tumour of a soft, transparent, and vascular character, with budding projections. They contain no milky juice, but a mucus with cylindrical cells. Besides the tumours showing structure identical with that of compound racemose glands, the follicles or ducts become dilated more or less, so as to form cysts; one or more of these may prevail, so as to give a cystic character to the whole (*cystic adenoma*). Besides the cysts arising in this way others may be formed by a breaking down of the intermediate tissue, especially if it happen to be mucous tissue. But such pure adenomata are comparatively rare; it is far more usual to find the glandular elements surrounded and separated by a new formation, which may be so much developed as to more or less entirely take away the glandular character of the growth; this interstitial tissue may either be fibrous, sarcous, or mucous, constituting *adeno-fibroma*, *adeno-sarcoma*, or *adeno-myxoma*, or more rarely it is cartilaginous or areolar; or it may present characters combining these or mediate between them. When the proportion of gland is small, there is doubt whether it is not part of the original gland-tissue persisting in the new substance. Thus, we see that in adenoma, &c., the relative augmentation of the cavities of ducts or follicles may make the tumour take the character of cyst, or the relative augmentation of the intermediate tissue may make it take the character of sarcoma, myxoma, or fibroma, so that it may become doubtful to which class we should refer the tumour. But a much more important ambiguity has been introduced because of the likeness of the microscopic structure of adenoma to that of carcinoma; this is so close that some observers class them together. We have, in speaking of the tumours of the breast, discussed this question, and concluded that the difficulty can only arise by too exclusively regarding the microscopic structure of the growths. Nay, that their likeness is rather in verbal description than in reality; for in adenoma the substance of the tissue is just that of gland-follicles, with regular epithelial lining and delicate fibrillar stroma between, while in carcinoma the epithelioid cells are not like gland-cells, but are of large size and various shapes, and have great nuclei with many nucleoli; the stroma, too, is generally different from that of adenoma, being composed of great spindle-cells, or more frequently of an areolar tissue highly charged with small roundish cells; but the chief difference is this, that the unaided senses can already detect the extension of the carcinoma to the surrounding tissue, and the invasion of the lymphatic glands, &c., which constitute its malignancy.

**Carcinoma.**—This name is now restricted to tumours whose histological structure is composed of an alveolar meshwork of fibroid tissue, the openings of which contain collections of epithelioid cells, filling up all the openings of the areolæ, and so composing a complementary epithelioid meshwork. Thin sections of such a structure show areolæ bounded by a fibroid substance, the epithelioid cells appearing within the areolæ lying close together without any intercellular substance. But a section of any secreting gland, such as the kidney, would show in like manner a fibroid stroma forming areolæ, in which epithelioid cells are lodged. These characters then are, so far, common to the structure of gland and carcinoma; we have already said that adenoma is the name given to a tumour which has the structure of gland, and hence adenoma and carcinoma, are, so far, alike.

But the difference between them is in the comparative perfection of pattern in the adenoma as contrasted with carcinoma. The structure of gland shows regular alveoli composed of a fibroid structure of areolar tissue, and containing epitheloid elements which are of uniform shape, and have small nuclei. But in carcinoma the alveoli are very irregular in form, and the substance composing their walls contains many spindle-shaped or rounded nucleated cells. Also the epithelioid contents show cells of very various sizes and shapes, with large nuclei and bright nucleoli, signs of active multiplying power. This cavernous arrangement of the stroma at once differentiates cancer from sarcoma.

These differences between adenoma and carcinoma are very important, because they correspond to a great difference in their history, carcinoma being malignant, so that it tends to spread in the surrounding tissues and to invade the glands and infect the great viscera remotely, while adenoma is a non-infectious growth.

Five leading types of carcinoma may at present be conveniently distinguished:

1st. Those in which the fibrous meshwork preponderates and the epithelioid contents are scanty, so that the growth is hard and firm—*Scirrhus* or hard carcinoma. Sometimes the epithelioid cells are not only scanty, but prone to perish early. Some use the term *Atrophic Carcinoma* for such tumours.

2nd. Those in which the fibrous meshwork is in smaller proportion, and the epithelial contents are plentiful, making large collections of cells, but with no evident approach in the form of these collections to the shapes of gland-acini, and no evident resemblance of the component cells, either to the columnar epithelium of mucous glands or the squamous epithelium of cuticle—*Soft* or *Medullary Carcinoma.* This kind occurs especially in the soft parenchymatous secreting glands, and the transformation of the glandular tubes or follicles to cancer-alveoli can be seen in all stages in the growing margin of the tumour, as, for example, where a renal tubule, containing a cast, lies beside two cancer-alveoli developed from neighbouring tubules.

3rd. A structure essentially such as that last described, but with this difference, that the epithelioid cells have a quantity of mucus between them, which is regarded as arising from a transformation of them. This change to mucus may be carried to such an extreme that scarcely any cellular elements are left, while the alveolar meshes in which the mucus is contained become very strikingly visible from their nakedness and the pellucidity of the mucus—*Colloid* or *Alveolar Carcinoma*. A common seat of this is the wall of the alimentary canal, where it may be traced arising from Lieberkühn's follicles.

4th. A structure in which the epithelial cells resemble squamous epithelium, and form masses which are very like the follicles of cutaneous glands, or occasionally like rudimentary hairs; the tubular and bulbous forms may, however, be seen ramifying like the lymphatic vessels of the skin, as if their form were moulded to the lymphatic plexus. In these cancers peculiar bodies are found, composed of flattened cells disposed concentrically so as to form a scaly-walled globe, whose appearance is like the section of an onion, or like a bird's nest; these are so large as often to be visible to the naked eye; when they are numerous and well characterised they are diagnostic. Some authors (Billroth) distinguish a variety of this cancer in which the stroma preponderates over the epithelial part, calling it scirrhus of the skin—*Squamous Epithelial Carcinoma.*

5th. A structure in which the epithelial cells resemble ordinary columnar epithelium, and the structure itself is quite like normal mucous membrane, in which it always primarily arises (alimentary canal, especially colon; uterus); the secondary formations which occasionally occur in these cases, in the liver especially, have the same structure, and thus a tissue like the glandular mucous membrane of the colon may be found in the liver—*Cylindrical Epithelial Carcinoma* or *Tubular Epithelioma.*

The fourth and fifth varieties are distinguished from the first three as epithelial cancers or epitheliomata. Some authors have used the term cancroid for the fourth variety, as though it were not completely cancerous. These are less likely to infect the viscera than the first two varieties, which are the most infectious of all tumours, though they are very far from being the only kinds of infectious tumours.

When in the first or second variety pigment appears within the cells the name *melano-carcinoma* is applied to the tumour. Other melanotic cancers consist of sarcoma with pigment in its cells; these are called *melano-sarcoma.*

It is thus seen that the prevailing opinion at the present day is that the sarcomatous tumours spring up in the connective tissue, whilst cancers and adenomas arise in epithelial structures. The stroma in which the epithelial elements are embedded puts on an incomplete glandular form; cancer is thus regarded by Virchow as an alveolar framework of connective tissue containing cells; the cells themselves having no specific character as once thought. Nor indeed is malignancy a special attribute of any growth. The same kind of growth may remain local for years or become metastatic.

**Lymphoma.**—The name lymphoma is given to such growths as have a microscopic structure like that of lymphatic glands; in particular, which have a finely reticular meshwork; connected with which are some fixed cells at tolerably regular intervals, not unlike the fixed cells of connective tissue, but generally larger. Within the meshes of this network are numerous cells, which resemble lymph-cells, and hence are also like pus-cells and white blood-cells. The proportion of network to the contained lymphoid cells is variable; sometimes the quantity of fibre is great, and the structure is then like lymph-gland tissue hardened by chronic inflammation. In other cases the proportion of cells becomes very large, while the network grows very delicate and open textured. The fixed stellate cells appear to multiply, and produce a progeny of the loose movable cells in the meshwork, as if infected by the latter. The whole mass then appears as fine filaments, making bold meshes, which are filled with round granular cells, like lymph-cells, but generally larger, and having a large nucleus and many bright nucleoli. These cells first appear to make up the whole substance, but they easily brush or wash out of the meshes, leaving the network very conspicuous. It will be noticed that the degree of structure here described is very rudimentary. Indeed, in sections of hardened ante-mortem blood-clot from within a vein, parts may be found which closely correspond to the description. So also tubercle in the more recently formed outer edge of it, where its texture is very like such portions of blood-clot, has the same texture, and hence it is by some classed with lymphoma. Scarlatinous tonsils and typhoid Peyer's patches likewise have lymphoma structure. However, the plan of structure is so meagre that it is not enough to form a bond of union between diseases clinically so remote from each other.

When found in the form of tumours, more properly so called, lymphoma generally takes its rise in the lymphatic glands; those of the neck are especially liable to it, then those of the abdomen and of the mediastinum. It is found in the alimentary canal, especially the small intestine and stomach, and in the spleen, liver, kidney, &c. Formations of a similar structure have been met with in various organs in leuchæmia, chiefly in the liver, in the form of small grains of a pale substance.

Lymphoma may prove malignant, that is, infectious to parts around, especially when the cellular elements are very numerous (in which case the tumour is called lympho-sarcoma by Virchow); it then corresponds to a part of what used to be included under the whole name medullary cancer, which, as formerly used, would include also soft sarcoma and soft carcinoma. Indeed, these tumours, when the cell-elements greatly preponderate, become very like each other, if not undistinguishable, as far as their mere structure is concerned. We have already spoken of the relation of lymphoma to lympho-sarcoma.

*Angioma*, or *Vascular Tumour.* When it is made up of capillaries it is called *telangiectasis;* when composed of spaces connected with the blood-vessels *cavernous angioma*, as has been described in the liver; angioma of the small lymphatic vessels is called *lymphangiectasis;* if made up of dilated spaces, intercommunicating, and filled with lymph, it is called *cavernous lymphangioma.*

*Teratomata*, or monstrosity tumours. These are made up of parts which might have existed in a monster, but are here irregularly thrown together and compressed into a tumour; they are therefore congenital. They may contain bone, cartilage, fibre, &c., and are frequently found on the sacrum and on the genital organs, but also on the head and other parts. *Dermoid cysts* constitute a variety of these tumours.

# GENERAL DISEASES

Having considered the structural changes in the various organs separately, we have in the majority of cases discovered in them the cause of death, or at least an insight into the nature of the malady which has led to it. There are cases, however, where no marked morbid appearances whatever may be found in the body, but where the symptoms are so defined that we cannot hesitate to declare that certain organs were especially disturbed and their functions overthrown. Of this class are such affections as tetanus, chorea, mania, which we have not hesitated to place under the head of "Nervous System." There are still other cases where morbid changes in the ergans are sufficiently well marked, but, at the same time, are too extensive to allow us to place our hand on any one part and say that is the seat whence all other changes arose; we are forced, then, to assign the primum mobile to the blood itself. Of this class are the fevers. There are also some chronic affections whose symptoms are characteristic enough to suggest names, but of whose pathology there is still some doubt, such as diabetes, myxœdema, Graves's disease, &c. Of this class we shall simply state what morbid conditions we discover.

Quite exceptionally we have to examine cases where we find no cause of death in the organs of the body; but, generally speaking, these cases have been only a short time in the hospital, and no opportunity has been afforded of studying the symptoms, the history which is given of any previous brief illness being most obscure. After excluding the question of poison we are obliged, in these instances, to have recourse to such conjectures as rheumatic hyperpyrexia, acute diabetes, and some other conditions to possibly account for death.

It is very obvious, therefore, that a simple demonstration of the morbid anotomy of particular parts is only one step towards a knowledge of pathology; while the study of their morbid associations as we find them grouped in actual cases raises us a step considerably higher, so that we believe a thorough acquaintance with the whole of the morbid processes, in any particular case, by enabling us to see how one morbid change is related to another, will lead us very close upon its true pathology. Our present means of investigation, however, will not allow us to demonstrate all the various pathological changes occurring in the body; for there are many, especially those in the nervous system, of which we are at present profoundly ignorant. It may be that some of these changes are of a chemical rather than an anatomical nature, affecting the composition of the juices rather than the forms of the solid parts, as appears to be the case in diabetes, &c.; and it may be that these ptomaines or animal alkaloids, like the poison strychnia, are of a nature related to the vital or nervous force, calling out the nervous force of one part of the body to effect the death of the rest without any anatomical alteration of either. Even when the morbid conditions are widest, we must not take them as ultimate facts, and think that we have arrived at the true pathology in them. For very often the great mass of disease which we find in a body is secondary, only representing the effects of causes that escape our observation, even when the proper connection of the appearances we discover is clearly traceable, which is not always the case. It is this tendency to lose sight of the causes and treat the effects as ultimate which has brought some odium on the science of morbid anatomy. We need not, however, be alarmed at studying it too much, for by a thorough knowledge of effects we must be in a better position to apprehend the nature of their causes. Without slight to the study of etiology and nosology, we may hold that the anatomical side of diseases offers us a view of them which we can realise and know, and upon which we can mutually compare our knowledge, while the sciences of the causes and symptoms of diseases are often vague or fragmentary, being too much mixed up with tradition and hearsay and ill-contrived experiment.

**General Appearance of a Body.**—We shall first of all draw attention to the external appearance of the body so far as it can assist us in forming an opinion as to the cause and mode of death. We may sometimes be required to inspect a body about which we know nothing, as we have constantly to do at this hospital; and therefore we should, in the first place, thoroughly examine the exterior before we begin to look inside. We should first notice that decomposition is sometimes very early in its appearance and sometimes very partial. One of its very earliest signs is often a formation of purple streaks along the courses of the superficial veins; whenever we see these streaks we may know that the inner surfaces of all the vessels, and the interior of the heart, and of the trachea, will be deep red, and we must never think this redness a sign of inflammation under such circumstances. The great parenchymatous organs, liver, spleen, kidneys, &c., will be dusky purplish, and their vessels, perhaps, will contain air. We can always tell such gas from the air of emphysema by its inflammability. These are states which denote blood decomposition, and that is all they are able to show; while they may render an opinion on the previous state of the viscera difficult or worthless. Sooner or later, they all set in, along with decomposition, in all cases, while the blood exuding from the vessels at the same time similarly stains the textures in the dependent parts of the body, into which the blood sinks by gravitation. Decomposition then soon declares itself, the abdomen turning green, and becoming distended with gas, the presence of which forces frothy fluid from the mouth and nose. In some cases the abdomen changes to a greenish colour before the blood decomposes. In these cases the decomposition is slower. Although there is no doubt that the reddening of the vessels, &c., which we have described, is due to post-mortem decomposition, yet it does not follow that the conditions of blood favouring such changes do not precede death; indeed, the especially frequent occurrence of these changes in febrile cases would indicate their dependence on a morbid state of the blood. They may often be observed to be strongly pronounced, when other bodies which have lain much longer in the same dead-house show no trace of them. The more advanced stages of decomposition, when greenness extends over the surface, is succeeded by blackness and giving way of the textures, which we can better observe in the dissecting-room.

In our survey of the body we should seek carefully for injuries, bruises, or the like, and never forget, in the absence of all other cause of death, to examine the cervical spine for fracture, or the larynx for a foreign body. When the body looks healthy, we may suspect an accidental death, or an acute disease, or one which has proved fatal suddenly; but if it be wasted, we at once discern that a long-standing ailment must have existed, and then it will be well to put the body through a course of physical examination. In chronic disease the emaciation varies in different affections; the most common cause of it is phthisis; but it is not so excessive as when death has occurred from abdominal affection, as of the stomach, mesentery, &c.; in phthisis the face retains a certain amount of plumpness, while in abdominal disease, where actual starvation takes place, the wasting is much more obvious, the temporal muscles are atrophied, and the fat removed from the orbits, so that the eyes will seldom close; the skin too is soft in phthisis, while it is often harsh in the other affections. We may notice too, with advantage, the condition of the hair in these wasted bodies, which always tells a tale of long-standing disease: instead of its presenting the curly crisp appearance of health, it is long or lanky; and this is especially observable in the beard and on the pubes; we may also notice that the hair has grown considerably on the chest in phthisical subjects. We then generally percuss the body, and if we find the chest dull on one side, we obtain a notion of phthisis; if with emaciation, the disease is chronic; if there be not much wasting, it is more acute, or acute upon old; further, if we open the mouth, and again strike the chest, we may often obtain the sound known as the *bruit de pot fêlé*, generally, but not certainly, indicating a cavity; sometimes we find the chest tympanitic, denoting a *pneumothorax*, and thus explaining the sudden death of our patient since we saw him last. In a case which occurred the other day, where the chest was dull and the body not wasted, and at the same time an herpetic eruption existed on the lip, the diagnosis was *pneumonia*, which turned out to be correct. In those dead of various chronic diseases we may often find that pleuritic effusion or pneumonia has ensued since the last time we were enabled to examine the patient alive. As a good example of the value of physical examination, we may mention a case which occurred some years ago, where the diagnosis made after death proved to be correct in every particular. A lad came one Wednesday morning to be admitted, and died in the taking-in room. Before hearing from his friends we proceeded to examine the body. On percussing the chest, the left side was found tympanitic, indicating a pneumothorax, but then the body was not sufficiently wasted for phthisis; and this sign suggested, therefore, an acute disease of the lung, which had ended in rupture of the tissue; as this, however, does not occur except in lobular abscess from pyæmia, a local cause was looked for as a source of purulent infection, when all that we discovered was a discharge from the ear, and on placing the fingers on the neck a general swelling was found, and an induration of the jugular vein; it became then tolerably clear that a chronic otitis, with diseased temporal bone, existed; that the lateral sinus had become involved; that phlebitis had extended down the jugular vein to the heart; and so the lung had been affected with abscesses, one of which had burst, and caused the boy's death by pneumothorax. On subsequently opening the body the morbid conditions were found true in every detail, and the whole history was exactly as suspected. Many similar instances we might mention, proving the value of physical examination.

Sometimes we have a person brought from the street who has died from *pulmonary hæmorrhage*. If the body be wasted, we suspect phthisis; if not, aneurysm. In *brain diseases*, the body too is often extremely wasted, but the emaciation, as far as we know, offers nothing characteristic, though, if paralysis or softening of the brain has been present, we may find rigidity or contraction of one particular limb, or the hands clenched, and the thumb drawn towards the palm, and generally a greater proportional wasting of the muscles than in other cases of emaciation; and the abdomen is often remarkably flattened or hollowed out, and we may find irregularity of the pupils. As regards this latter condition, we would say it is only in exceptional cases that anything unnatural is seen in the state of the pupils after death; the rule being that they are symmetrical and of ordinary size, the tendency being at death for the two antagonistic sets of muscles of the iris to neutralise one another. In a word, we might say that the state of the pupil affords no indication of the mode of death. There are various other conditions of the body to be observed; thus, if it be not wasted, and the circumstances of the death suggest some acute affection, we may then look to the skin, and sometimes find a petechial mulberry rash covering it, denoting *typhus fever*. In typhoid fever no rash would be present, except perhaps a slight purpuric mottling of the legs. If there be an unhealthy sore on the body, and especially if a bone be exposed, we may suspect pyæmia, and the suspicion is strengthened if the body have a yellowish tinge.

In acute *abdominal diseases* the distended tympanitic abdomen may suggest the idea of *peritonitis*, particularly if the countenance express what we call abdominal anxiety, still shown after death by the ghastly visage with sunken eye, &c. In cancer of the stomach the wasting is the most extreme that we meet with ; various tumours may be felt after death as during life, and attempts at diagnosis should be again made.

If the body be dropsical, this denotes, in by far the majority of cases, some visceral disease (it may accompany mere anæmia and cachexia) ; if all parts are affected, it shows that the cause is not a local one, but is to be sought in the blood ; and we, therefore, if we see the arms, face, scrotum, &c., swollen, pronounce in favour of renal disease. If the lower parts of the body only be dropsical we suppose the dropsy to be due to a mechanical cause in the chest, and then comes the question, Is this a pulmonary or a cardiac obstruction ? If there be great lividity of the face we may suspect bronchitis ; but if this be less, and if the skin of the upper part of the body be yellowish, denoting hepatic engorgement, we may be pretty sure that the person has died of morbus cordis. The appearances in such a case are so well marked that we may almost always recognise a cardiac case before the body is opened. If the abdomen alone be dropsical, as in *ascites*, a liver disease may be suspected, or a chronic peritoneal affection. If the legs be swollen, or only one leg, it may be well to look for a plugging of the femoral vein with ante-mortem clot.

Besides noticing emaciation we should observe whether there be a superabundance of *fat*, for this denotes a morbid condition of the system which accounts for many deaths taking place in persons after slight injuries or surgical operations, such as that for the relief of hernia. We may often notice in persons advanced in age a large amount of fat beneath the integument of the abdomen, as also a large quantity within its cavity, occupying the mesentery, omentum, region of the kidneys, &c. ; this is one of the conditions of age ; but we may occasionally observe the same in younger persons, especially those who have been much addicted to intemperance, and more particularly to beer-drinking, for spirits unless indulged in very largely and for a short time, so as to prove rapidly fatal, tend to wasting of the body. We also get accustomed by experience to draw conclusions from the quality of the fat, there being a firm, whitish, adipose tissue, which denotes health, while there is a yellow oily form, which may be looked upon as morbid. We should also examine the eye for *arcus senilis*, which generally indicates senile or fatty changes throughout the body. The *muscular tissues* should also be noticed ; there is the large red muscle denoting a healthy state, and the atrophied pale muscle showing at least a long-continued inactivity, with perhaps also some degree of fatty degeneration.

*Rigor mortis* should also be looked for; if present it shows that death has not long occurred; but it varies with the mode of death and state of the weather. It may continue under favorable circumstances as long as ten days. It generally begins within five or six, some say eight hours after death; we have repeatedly found it distinctly pronounced, though as yet slight, in the sixth hour. It is said that in persons who have died with absolute suddenness by shots through the heart, &c., the rigidity comes on at once and persists. It is usually affirmed that rigor mortis commences in the muscles of the jaws, and extends downward to the neck, trunk, arms, and legs. This order, however, is by no means absolute; we have often found rigor mortis present already in the legs when there was none in the arms. The jaw is, however, the part usually first affected. The strength of rigor mortis in any part of the body is in proportion to the power of the muscles that act on the joints there. But in different persons it varies in strength, when at its height, with age, and with the cause of death. Thus it is scarcely noticeable in infants, and is feeble after prolonged and wasting diseases; it is always most marked in the bodies of robust persons who have died by some rapid disease, such as cholera, &c. Of the cause of the change little is known; it has lately been ascribed by Kühne to coagulation of the myosine, with loss of elasticity and contractility of the muscular fibre, this at the same time becoming acid. As the acidity passes off rigor mortis disappears, and an alkaline condition and fermentation ensues.*

We shall now proceed to give a description of the morbid changes found in the organs of the body in those cases which bear some of the more general names just mentioned.

**Pyæmia.**—The cause of death after great wounds or extensive sores, or gangrene, was formerly little inquired into; it was ascribed to shock, fever, or such like vague considerations. But when post-mortem examinations were made, it was found that internal inflammations are present under these circumstances, and often are evidently sufficient to produce a fatal result. These inflammations, however, are of a peculiar kind. Their most marked feature is a tendency to the rapid formation of pus; hence it was natural to think that the pus had been taken up from the wound or sore, and lodged in the internal parts where it was found; and thus the earliest and simplest notion of pyæmia arose. But it soon became evident that the pus in the internal parts is formed by inflammation, and is not simply conveyed there; for the inflammatory process is evident around and in the puriform collection.

The idea and word *metastasis*, or transference of a diseased process to a new seat, here lay ready for employment; and, indeed, this vague term still serves a useful purpose in expressing the shifting of a diseased action from one place to another; still, such vague ideas, if accepted as sufficient, repress inquiry, and we must, here as elsewhere, take the facts of the disease, and the results of experimental inquiry, to throw light on its actual nature.

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* See paper on "Cooling of the Dead Body," by Dr. Alfred Taylor and Dr. Wilks, 'Guy's Hospital Reports,' vol. ix, 2nd series.

Such, indeed, has been the course pursued, and now we are in this position—that we have two distinct departments of facts bearing on the question of pyæmia; the facts from human morbid anatomy, and those from experiment on animals. To take the latter first, many distinguished observers have studied the effects of injecting putrid matter into the veins or the cellular tissue of dogs and other creatures; the result is the literature of *septicæmia;* but the bearing of this on our knowledge of pyæmia is not as yet very direct, for by such experiments we do not generally produce those suppurations that are characteristic of pyæmia as met with in the post-mortem room. The animals experimented on recover either with or without grave symptoms; or die generally with diarrhœa and collapse, when the alimentary canal shows on inspection signs of inflammation after death, and there are congestions, ecchymoses, or inflammations in the lungs and other organs, that is, such signs as prove a poisoned state of the blood.

With some the term septicæmia is limited to this class of case where the blood is poisoned and followed either by rapid recovery or where, if death takes place, no local processes are found. Pyæmia is an affection of a more malignant character and ends fatally by the production of pathological changes in various organs.

It has been said by Dr. Burdon Sanderson that the poison of pyæmia increases in intensity of action in animals successively inoculated with it one from the other; so that one animal, inoculated with matter from the peritoneum of a former victim just dead of disease caused by inoculation with bacteriferous septic matter will suffer more violently and die quicker than the first victim. And a third animal, inoculated from the second, will die yet quicker. So that it appeared to Dr. Sanderson that the poison was capable of intensification by a sort of cultivation.

These experiments were repeated by Dr. Goodhart,* but not with the same results. He found bacilli and micrococci in the wounds and blood of those who died of pyæmia, but they were not always present, nor in any way characteristic. This was also the conclusion arrived at by the Committee appointed by the Pathological Society.

In our experience of deaths from wounds, sores, &c., we have not met with any class of cases that at all closely correspond to these results of experiment by showing enteric disorder, collapse, and quasi-choleraic phenomena. So that we think it is better at present to regard the history of the experiments on animals that we have alluded to as a distinct branch of the history of septic infection, whose bearing on clinical pyæmia is as yet a problem requiring solution.

But if we take the morbid appearances in the human body after death from grave wounds and sores, these form a very definite series, one to which the term *pyæmia* properly applies. We have already described the appearance of the changes in each individual organ, and will now only consider their distribution in the body and their general features, so far as will serve to give us a view of the nature of the disease. The most frequent seat of the abscesses is in the lungs, and as these organs receive the blood from all parts of the body, this at first favours the idea of a simple transfer of the pus or of some cause of pus, in the blood, from the sore to the lungs. In other cases the liver is especially affected, and it is interesting to notice that in these cases the sore is generally in the alimentary canal, from which the liver draws its blood. These two facts raise a very strong presumption that the cause of the pyæmic suppuration in the lung and liver is the introduction of pus, or of a pyogenic something, into these organs from the sore. The proof of this is rendered absolute in some cases by our finding, in the pulmonary artery in the lung, or in the portal vein in the liver, a portion of purulent clot, while the veins of the sore contain similar clot; and in all cases the peculiarly strict circumscription of the pyæmic inflammatory affection in the lung or liver corresponds suggestively with the like limitation of the distribution of individual branches of blood-vessels in the parenchyma of the organs, thus indicating a dependency of the local disease on some occurrence proper to the vessel.

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* 'Guy's Hospital Reports,' vol. xx, 1875.

In some cases no doubt can exist that the lung or liver is thrown into inflammation by the advent of a purulent embolus actually found in the vessels, and thus we have a sufficient explanation of the suppurations in those cases of pyæmia. They arise from embolism, and some would therefore speak of *embolic pyæmia.* Others, especially in Germany, go so far as to hold that all secondary abscesses arise thus from embolism, and so make all pyæmic suppuration a department of embolism. This we shall see at least the following reasons to think is not correct.

Thus, if we observe the position of the pyæmic abscesses in the lung, we find that a very great part of them are limited to those parts where emboli are least likely to alight. Again, although the lung is more generally affected in pyæmia than other organs, yet sometimes we find pyæmic abscesses in the liver, &c., as after injuries to the head, when there are no abscesses in the lung. Yet any pyogenic matter must get through the lung on its way from the head to the liver, and one knows no mechanical reason why matters which have passed the pulmonary should stick in the hepatic capillaries. Again, it is yet more frequent to meet with cases, generally more chronic in their course, where the abscesses all form in the external parts of the body, that is, in the teguments, in the muscular tissue, joints, &c. Such cases may recover through the freedom of the viscera from implication. The affection of the joints is often so marked as to justify the term *rheumatoid pyæmia* as applicable to these; and when we consider that through the medium of gonorrhœal rheumatism and scarlatinal rheumatism these cases are linked to rheumatic forms of arthritis, we find ourselves getting a long way off the embolic pathology we have just described, because the regions attacked are not such as are exposed to embolism; and when, again, we believe that cases of pyæmia occur entirely without any kind of wound or sore—that is, where the most careful search showed the absence of any primary sore—then we find ourselves in another way deprived of the embolic theory, because there is no showing any source for the emboli. Lastly, we must remember that some kinds of pyæmia appear to elect proper seats of secondary suppuration, especially the pyæmia which follows suppurative periostitis, which produces abscesses in the heart and kidneys, usually without affection of the liver; while, on the other hand, injuries of the cranium, affecting its sinuses, often produce abscess of the liver. Again, pyæmia, after typhoid tends to affect the salivary glands; that after scarlatina to affect the joints.

The above facts, taken severally, show that embolism is not the only cause of pyæmic suppuration; taken together, they go far to reduce embolism to the dimensions of one incident, which, among others, determines the localisation of pyæmia on special points.

The report of the Pathological Society showed that visceral abscesses occurred without any evidence of thrombosis or embolism, and, on the other hand, thrombosis occurred without any secondary abscesses. In some cases the two were found together.

Many facts go to show that an impure state of the blood produces greater effects where there is stagnation of it. This is plainly indicated by the position of the abscesses in the dependent part of the lung, but the same truth is also shown by the suppuration of the veins in which thrombi have formed. This we have seen in several marked instances, especially about the prostatic plexus, or behind the valves of the great veins, or in the cranial sinuses. Here the explanation is almost as clear as that of embolism, for it is certain that when the blood is impure the impurity will be increased by accumulation where it ceases to circulate, since the effect of circulation is to bring the blood under the action of purifying organs. This we see is so certain that the explanation of the occurrence of suppuration in dependent and stagnant situations is almost as secure as that of embolism; and it is quite possible that the two conditions may coexist, so that when small emboli are sown broadcast in the vessels of the lungs, those which lodge where the blood is purified freely in the upper part of the lung may not be able to excite suppuration, while those which stay in the stagnant lower lobes, already œdematous and scarcely expanding at all, suffice to arouse the inflammatory process.

From these considerations it would seem that if we may use the term embolic pyæmia as signifying cases where embolism is proved to occur, then we may also use the term *stagnative pyæmia* to indicate the formation of abscesses by the stagnation of poisonous matter within coagula in the affected part. The other cases we have alluded to, where the joints, kidney, or liver, are especially selected, are not at present susceptible of more than such hypothetic suggestions as that the poison has a natural affinity for the parts that are inflamed. For when we neither have the embolus nor stagnation to explain the local affection, as in cases of the special disorder of the joints in rheumatoid pyæmia, the glands after fevers, the kidney and heart after suppurative periostitis, then we are obliged to assume an attraction between the part affected and the pyogenic matters in the blood. Of course, this is unsatisfactory when compared with the clearness with which we can follow the embolus in its course or anticipate the effects of the stagnation. But it is the best that we can do at present, to consider the fixation of pyæmia in the joints as analogous to the fixation of smallpox in the skin and of typhoid in the intestine.

After this general survey of the indications of its morbid anatomy, we must regard pyæmia as made up of blood disease and local inflammations. We can trace several ways in which the blood disease and local inflammations are related to each other.

First of all, the general disease may greatly predominate so that the blood becomes unviable, and death occurs with signs of blood-poisoning, revealed during life in the form of high fever and rapid fatal exhaustion, and after death by softening of the great viscera and extravasation of blood and ecchymoses within the tissues, no local suppuration being found. Also we must remember that we meet with cases of pyæmia of a most characteristic kind without discovering any kind of sore whatever; at least we have in several instances searched with the utmost care through all parts of the body and found no primary disease.

But in many cases such pyæmia as this is traceable to the contagion of ordinary fevers, such as scarlatina and diphtheria. It is especially to the pyæmia of the puerperal state that this applies; the results of Dr. Hicks's observations showed that in by far the majority of the cases of puerperal fever the patients had been exposed to contagion of some known fever. The existence of a proper pyæmic contagion is a matter much contested at present, and there is a further contest among those who would admit such a contagion upon the question whether the poison enters by the wound or by the natural surfaces of the patient. In considering the question we cannot disregard the antiseptic treatment, nor the fact that some kinds of wounds are more infective than others; those, for instance, connected with bone.*

In another kind of pyæmia the wound or sore produces a morbid matter which is absorbed and so poisons the blood. It is probable that the pyrexia of surgical fever is principally caused by the absorption of the products of the wound, so that this kind of pyæmia is allied to common surgical fever. The prevention of putrefactive changes in the discharges of the wound averts the danger of pyæmia from this cause, and the success of the antiseptic method shows sufficiently the importance of this source of blood-poisoning.

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* See paper on "Pyæmia," 'Guy's Hospital Reports,' vol. vii, 3rd series.

In some cases that go by the general name pyæmia the whole affection is confined to the formation of purulent thrombi in the veins, and the conveyance of these to the lungs as emboli, where they set up secondary suppuration. Such a process is too simple and clear to need further remark.

In these three divisions, each of which is represented by some unquestionable case, we have three kinds of pyæmia, kinds, that is, in the sense that they are disorders of an entirely distinct nature, but we would wish to observe that they are not different kinds in the sense of being incompatible or necessarily alternative. Far otherwise, they may all coexist; it is quite possible that a wounded person may breathe the contagion of a fever, or some other foul matter, his blood thus becoming poisoned; and that as a consequence of this, the secretions of his wound may be more putrescible, thus producing noxious matter that is absorbed in the blood, adding another depraving element, whose tendency is to set up remotely the condition present in the wound; and, again, that the low state to which nutrition is reduced may render the veins vulnerable, so that these inflame and produce purulent thrombosis, from which embolism conveys portions to start abscesses elsewhere. And although we do not prefer the lumping together of distinct alternatives into one view, yet it is probable that, in the genesis of pyæmia, this association of distinct elements in its causation actually does occur, and that pyæmia is first started by a depressed state of the blood, and that this reacts on the wound, causing in itself and in the vessels around the putrefactive and phlebitic changes which lead to these internal suppurations that give to pyæmia its distinctive characters and its name.

The view we have given, then, may be thus summed up:—Simple embolism, purulent absorption or general fever, due to contagion, severally form distinct varieties of what is called pyæmia; but in many cases, perhaps the majority, all these conditions exist together.

We have said little about bacteria, because even if pyæmia be of a specific character the presence of microbes would not alter the general description of the disease which we have given.

**Puerperal Fever.**—We have already described the inflammatory processes in the uterus and its appendages which may follow parturition; and, when speaking of these, remarked that in proportion as fever following parturition was ascribable to simple inflammation about the uterus, in such proportion is it unnecessary to suppose the existence of any specific puerperal fever. But in a considerable division of such fevers the local inflammation was trifling or absent, and the disease comes under the pathology of pyæmia. When speaking just now of pyæmia, we mentioned Dr. Hicks's observations on puerperal fever, in which he showed the frequency of contagion from ordinary fevers; indeed, it appears that any general distinction between puerperal fever and pyæmia rests only in the relative frequency of contamination by fever poisons, which is much greater in the puerperal cases.

For the rest, inspections of bodies dead of puerperal fever will offer examples of all the varieties of pyæmic accident we have before described.

**Typhus Fever.**—The characters of typhus during life are those of a simple fever, generally without complication; and after death we generally find no local anatomical disease. And yet the conditions of the blood and viscera give evidence of great changes, so that although there is no definite morbid anatomy to typhus, yet we think that an experienced observer would be able to refer the death to this disease by post-mortem examination; unless decomposition had already set in, when this would be impossible, because the changes in the blood and viscera in typhus are almost identical with those brought about by incipient putrefaction. Decomposition in typhus commences early. The petechial part of the mulberry rash persists in the skin, and on examining the viscera they are found to be soft and full of dark fluid blood. The heart is flabby and lacerable, its fibres often granular under the microscope; this condition of the heart has been described as a special disease in fever by Louis and others; it is found in most cases, but is not absolutely constant; the heart may appear healthy, and well contracted, and sometimes Zenker's waxy degeneration has been found in it.

The brain is healthy in typhus, though it has been asserted that the cerebral capillaries contain clumps of white corpuscles to account for the delirium, &c., in fever; this, however, we cannot ourselves support, the appearances figured being such as we have seen in ordinary conditions. It is well to remember Dr. Murchison's suggestion that epidemic cerebro-spinal meningitis is typhus complicated with inflammation of the brain; still, at present this is only a surmise, and if now meningitis were found in any case of typhus, that case would be regarded as one of "cerebro-spinal meningitis." The lungs frequently show bronchitis, and they are congested with dark blood at their dependent parts. Occasionally we find pneumonia, generally in the lobular form, in the lower lobes. Lobar pneumonia is sometimes found but much more rarely than in typhoid. The alimentary canal may contain blood from purpuric extravasation; the walls are generally quite healthy, but the solitary and agminate glands may be a little enlarged, and the latter may present that dotted appearance which has been compared to a shaven beard. Occasionally dysenteric or diphtheritic inflammation complicates typhus, but this is exceptional. There may be gangrenous stomatitis or parotid suppuration in some cases. The liver is soft and the section shows a dull clay-like lustre, which is almost characteristic. The spleen is large, and very soft, its pulp easily washed away. The blood is described as containing an excess of white corpuscles, but this is not very evident. The kidneys are large and coarse, and the epithelium cloudy with granules, or disintegrating. Dr. Murchison has pointed out that after death by convulsions in typhus the kidneys are exceedingly large, and the urine albuminous; he has shown that, at the same time, urea is present in the ventricular fluid of the brain; urea may be found there, however, when no convulsions have occurred. Thrombosis of the femoral veins, with white leg, may occur, but it is not so frequent as in typhoid; it generally affects the left leg; fatal embolism of the pulmonary artery may result from it. In typhus, too, the arteries may become obstructed, sometimes evidently through embolism with antemortem clots from the heart; at other times no such clots can be found in the heart, and then some believe that the clots form in the arteries, a view which may occasionally apply, but we suggest, as more probable, the supposition that such clots had formed in the heart, and were thrown on into the vessel while yet recent. The extremities, nose, ears, &c., may be gangrenous; it is not yet settled whether this gangrene arises through obstruction of the arteries. Grave bed-sores may arise and extend enormously, causing necrosis of the trochanters or sacrum. The bladder may inflame, through neglect of its over-distension, or through careless use of the catheter.

**Typhoid or Enteric Fever.**—We have already fully described the changes in the intestines which characterise enteric fever; and the dangers of peritonitis, perforation, or hæmorrhages, which accompany these changes. As we then remarked, these intestinal ulcerations are occasionally accompanied by so little fever that fatal perforation occurs while people are at their work; but generally the patient undergoes the "typhoid state," and the organs of the body are found presenting the gorged and softened condition we described under typhus. It is remarkable, however, that the brain is usually firm. Otherwise there is often nothing to distinguish the general state of the viscera from that found in typhus. The heart is here, too, soft, and its fibres rather granular; or we may meet with Zenker's change, a condition in which portions of the heart are found too solid; the fibres there are easily torn, while the cut surface glistens; the muscular fibres present microscopically a peculiar waxy change. This, which also occurs in hyperpyrexia, has already been described under "Muscle."

We have sometimes found the larynx affected by deep ulcer at the root of the vocal cords. This has already been described. The air-passages are very apt to be inflamed, and often importantly so. The inflammation may extend into the lung, producing fatal pneumonia; this then is found in the form of lobular patches, in the dependent parts of the lungs. These patches are around the ends of the bronchial tubes, thus indicating that the source of the irritation to the lung comes down the bronchus. The form of pneumonia here alluded to is very frequent in fevers, and often is the immediate cause of death; it is sometimes called *hypostatic pneumonia.* We occasionally, but rarely, meet with a large tract of hepatization of the lung, as in so-called lobar pneumonia; and sometimes even gangrenous patches in the lungs.

We may find stomatitis, follicular or ulcerative, also suppuration of the salivary glands. The liver has the same character as we have described under typhus; it is soft, and its section has a dull appearance, like cut yellow clay; the microscope shows the cells granular and indistinct in outline. Some consider these appearances to prove the presence of early hepatitis, but there is no lymph on the surface of the liver to support this view. It is remarkable that the extensive intestinal ulcerations of typhoid very rarely lead to abscess of the liver. The gall-bladder, however, is sometimes said to be inflamed, and has even been found perforated; we ourselves have never seen this. The spleen is large and may be dark, and tolerably firm in the early stages, as felt during life; but at post-mortem it nearly always appears soft, and full of pulp. It is loaded with blood, and there is formation of an excess of splenic elements; such soft spleens are said sometimes to burst. The voluntary muscles, especially the abdominal rectus, diaphragm, adductors of the thigh, and the tongue, sometimes show the change discovered by Zenker. These muscles may be ruptured, either in consequence of that change, or without it through violent exertions in delirium when the patient is carelessly watched. Grave bed-sores or gangrene may occur as in typhus; obstruction of the femoral vein, and consequent white leg, is more frequent than in that disease. The eruption in typhoid fever disappears after death. As sequelæ of typhoid fever we occasionally meet with periostitis and pyæmia, and at a later period more rare disorders, as paraplegia.

**Relapsing Fever.**—The conditions observed in relapsing fever, with the exception of frequent presence of jaundice in this disease, do not differ from those seen in typhus. This fever is rarely fatal. So we have no experience of any post-mortem appearances. The most remarkable feature of the disease is the presence of spiral bacteria in the blood. These are long, spiral, flexible filaments, always in movement. The parasite is called "spirochæta."

**Intermittent and Remittent Fevers.**—Besides congestions and softening of the organs in acute, and anæmia and œdema or more general dropsy in chronic stages, there are other more characteristic lesions associated with these fevers; they go by the general name *melanæmia.* The blood is found to contain particles of pigment, and, as the consequence of this, the pale parts of the body may be of a dusky tinge, but the spleen, liver, and brain suffer especially. In these organs the pigment is found within and about the vessels, as though it had lodged in and obstructed them, leading sometimes to minute extravasations of blood; for particulars of the appearance, we must refer you to the melanic state of the several organs.

**Yellow Fever** has never come under our observation. From the descriptions of authors, it appears there is nothing characteristic, except the jaundice and the hæmorrhages; but the œsophagus and stomach may present considerable erosions and ecchymoses.

**Scarlet Fever.**—The morbid anatomy of this disease has been more especially investigated by Klein and Crooke. The specific microbe which has been discovered has been named by the former *Streptococcus scarlatinæ.* During life the whole of the skin is congested, and new products may be found within it on examination. In like manner the mucous membranes are found in an intense state of hyperæmia or pouring out exudations. The stomach, for example, has its mucous membrane swollen or mammillated and covered with secretion. An examination by the microscope shows a production of lymph-cells in the tissue as well as in the gland-tubules. At a later stage Fenwick has shown these tubes filled with granular and fatty matter, with some interstitial infiltration. In fact, the observers mentioned would say that in scarlatina there exists a distinct catarrhal gastritis. The throat also is a part especially affected, an infiltration taking place into the follicles of the mucous membrane and into the tonsils; and with the throat the neighbouring lymphatic glands are also involved, Klein finding within them large nucleated cells. In the intestine a follicular change also occurs, together with an infiltration of the glands; sometimes Peyer's patches are so much enlarged that they resemble the early stage of typhoid. The spleen is large, with an increase in size of the Malpighian bodies. The liver, according to Klein, shows an interstitial proliferation, and he suggests that this might sometimes be the starting-point for the cirrhosis occasionally met with in children. The kidney shows more than any organ the most marked changes. In the first instance it is hyperæmic and sometimes has hæmorrhagic effusions within it. As already mentioned, it is affirmed by Klebs that the peculiar form of inflammation in scarlatina is a glomerular nephritis; this statement Klein confirms, but says that he has also found an infiltration of cells in the interstitial tissue, and also a proliferation of the walls of the blood-vessels, which assume a hyaline condition. He regards the inflammation as an interstitial nephritis. Crooke confirms this germination of nuclei in the capillary walls, but says at the same time we may find an intratubular inflammation characterised by a cloudy swelling of the renal epithelium, and, subsequently, the production of hyaline cylinders and blood in the tubules.

**Measles.**—Both bacteria and micrococci have been found in the blood and in the nasal mucus; also peculiar shining granules in the skin, which have been regarded as specific in character. In fatal cases the bronchitis has usually terminated in a broncho-pneumonia; other organs present no peculiar appearances. As sequelæ of measles, we find occasionally brain affections as well as spinal, the latter showing themselves as paraplegia, also enlargement of the bronchial glands, which may end in caseation and bronchial phthisis.

**Diphtheria.**—In acutely fatal cases we usually find membrane on the pharynx, larynx, and bronchi; and, frequently, a lobular pneumonia. The submaxillary and parotid glands may be affected as well as the lymphatic. The kidneys, which have frequently produced albuminous urine, are congested, and show both a glomerular and tubular nephritis. It has been surmised that there might be a primary diphtheritic nephritis, this being indeed the only way in which the disease showed itself. There may be found in other organs ecchymoses but no structural changes. At a later period, if death occurs through anæmia, no very obvious organic changes are seen. Nor are there usually any if death has occurred by paralysis; some regarding this as altogether reflex, whilst other observers have described changes in the anterior cornu and motor nerves, believing that a neuritis has proceeded from the throat to the spinal centres.

**Malignant Pustule or Anthrax.**—This may be communicated to man from horned cattle who are suffering from "milzbrand," through the instrumentality of a very characteristic microbe called *Bacillus anthracis.* This parasite is remarkable for its length, is often jointed, and the spores within it well seen. The anthrax may also be introduced from the hides of cattle who have died of the disease, and consequently many cases of it are seen amongst the tanners in this neighbourhood. The same disease also may be taken from the inhalation of the dust of the wool of sheep who have suffered from a similar malady. This is known as the Bradford or *wool-sorters' disease*, and is rapidly fatal without the appearance of any external boils. We must refer to "Skin" for an account of the character of the pustule or boil, which is produced by inoculation. In the cases of infection through the air, which are rapidly fatal, the post-mortem appearances are only those which are found in fevers and cases of blood-poisoning; but in the fatal cases of external anthrax which occur at Guy's some very remarkable pathological conditions are found. The stomach has its mucous membrane swollen, and upon it some large black sloughs. The duodenum and jejunum also have anthrax growths, black and doughy, and around them a remarkable translucent œdematous swelling. The spleen is soft and diffluent; hæmorrhage may have occurred into the lungs, and ecchymosis of the brain and spinal cord, as well as of the heart, may be present.

**Glanders and Farcy** are names given to two varieties of *Equinia;*

the virus producing these varieties being identical, but in glanders it develops its effects chiefly on the nasal mucous membrane, while in farcy the skin and subcutaneous tissues suffer most. In the former case we find a large quantity of fœtid, tenacious mucus filling and running from the cavities of the nose, while the walls of the nares show ulcers, perhaps penetrating and destroying the bones, the earlier stages of the ulcers being in the form of tubercles or knots in the congested and swollen mucous membrane. If the case be acute the face is much swollen, the appearance resembling erysipelas, and, as seen in our models, bullæ may appear on the skin.

But the affection of the skin usually puts on the appearance of pustules, furuncles, and carbuncles; the early stages of these are in the form of vesicles resembling varicella or small red patches, which swell, and soften as they swell, until little abscesses are found raising the cuticle; they do not, however, arise as collections between the true skin and cuticle, but by infiltration and softening of the true skin itself. The process shows itself, microscopically, as a formation of small delicate cells in the areola of the cutis; these increase in numbers, and enlarge, until the whole affected spot appears made up of such cells, which are compared by Virchow to the tissue of granulations, so that he includes glanders in the class of tumours which he calls "*granuloma.*" In the further progress of the abscesses blood often becomes mixed with their contents; they open, and form ill-conditioned sores under blackish crusts. The lymphatic glands are apt to be affected; chains of glands may enlarge, forming the so-called "farcy buds," as in horses. The cause of death may be, in chronic cases, a slow exhaustion by the discharging sores; the disease appearing in the form of slow chronic ulcers, which may escape diagnosis. But in the acute form the interior of the body shows the condition of a very severe pyæmia. In the case of a man who was bitten by a horse, and was thought to have glanders, the post-mortem appearances were those simply of pyæmia, but in other cases we have found the muscles of the extremities stuffed, if we may so speak, with abscesses of a size from peas to chestnuts, while the lungs were so full of similar abscesses that nearly half the whole tissue was taken up by them. The kidneys, heart, liver, and joints were similarly affected. During life albuminuria is frequently present. Klein describes the presence of a bacillus found both in man and the horse, which resembles in form and size the tubercle bacillus.

**Cholera.**—We have examined many bodies of those who have died of cholera; the following were the appearances :—As a rule, the viscera showed no signs of organic disease, although repeatedly we sometimes found old long-standing morbid conditions, such as ulcerated intestine, degenerated kidneys, chronic endocarditis, &c.; these, however, were exceptional. The majority of persons attacked were healthy. In rapidly fatal cases, dying in a state of collapse, the temperature often rises after death, and the muscles may twitch for a time; the skin is livid, especially in the dependent parts, and the face has a ghastly sunken appearance; rigor mortis is generally unusually strong. The brain is natural, or shrunken a little, having then an excess of subarachnoid liquid. The lungs are in a peculiar state; they are of small size, and have a collapsed look, being dark and flabby; they are generally very light in weight. Thus, in one rapid case, that of a strong man, we found the weight to be but twelve ounces for the right lung and eleven ounces for the left. On section their texture is dry, the great vessels containing a moderate quantity of dark liquid blood. To understand their shrunken collapsed state, we must open the bronchia; we shall find them curiously dry within; now, remembering that what prevents collapse of lungs under ordinary circumstances is the quantity of the mucus in the tubes, preventing the exit of air, we see how the empty and dry tubes in cholera will permit of the lungs collapsing according to their natural tendency. The blood in the great vessels is dark and thick, from the loss of water. We see no exudations on the pleura. We have never observed the pulmonary artery unusually full of blood; in one or two instances we have seen embolic patches in the lung; the heart is generally contracted closely and firmly, and consequently its cavities, especially those on the left side, are closed and empty; on the thinner right side, the closure is less complete. There is an accumulation of blood in the great veins; the blood in the heart often contains white clots, or it is gelatinous, or tarry, dark, and thick.

The gall-bladder is distended with bile, and the liver itself appears healthy, but is flabby, and rather dry on section.

The spleen is flabby, perhaps rather tough; sometimes, however, it is soft; it is notably dry on section, and often small.

The appearance of the alimentary canal is peculiar, and almost characteristic of the disease. The stomach has its mucous membrane swollen and softened, the secreting tubes being filled with cell elements. The intestines lie in a very small space, and they are almost always quite devoid of gas, instead of, as is usual, being more or less distended with it; hence they have no disposition to uncoil or separate themselves, but form a flabby compact mass, feeling doughy and flabby, and pliable to the touch. They have a peculiar rosy pink colour, because the foul gases being absent, the usual staining of the blood with sulphuretted hydrogen does not occur.

Their surface exudes a tenacious mucoid secretion, not generally described, and this may be owing to its not being always present; it is not, however, uncommon, as may be best observed by separating two coils of the bowel, when a viscid mucoid string is drawn out between them. The secretion is hardly like that of recent inflammation, as the exudation is more like mucus, and is more sticky than the greasy sero-albuminous fluid of ordinary inflammation. The internal mucous surface of the intestines has a sodden appearance;

a white creamy secretion covers it, which, when mixed with a large amount of fluid, constitutes the rice-water evacuations.

The amount of fluid is variable in different cases; there are generally no natural fæces present, and there is usually no tint of bile in the contents.

The enlargement of the intestinal glands is often very remarkable; affecting sometimes, but not always, Brunner's glands in the duodenum, generally Peyer's glands in the ileum, and most constantly or universally the solitary glands both of small and large intestine. The two latter kinds of glands are sometimes so large that they give a resemblance to the early stages of typhoid fever. We might here remark that the condition of intestine described is not much unlike that found in cases of severe poisoning by large doses of arsenic, a fact important to remember when the symptoms of the latter during life also resemble those of cholera. The kidneys are generally soft, and of a bright red colour, weighing often rather less than their usual weight; sometimes, however, they are dark and congested, and often a slight change may be found in the epithelium. The urinary bladder is contracted and empty.

In the bodies of those who have died during the consecutive fever the characteristic appearances of the collapsed stage just mentioned no longer exist. Gas is now found in the intestines, and hence the peculiar colour and feel we have spoken of are gone, and the interior is found to contain bilious matter, though the glands may still be seen large. The kidneys are generally diseased, and in some cases have presented very much the appearance found in those who have died of acute scarlatinal dropsy; the organs are large and congested, weighing sometimes fifteen or sixteen ounces; the microscope shows the tubules gorged with a granular secreted matter, and very probably their pathological condition is similar to that of the scarlatinal kidney, as the symptoms of the two during life also accord. We have seen some cases of cystitis and suppuration of the kidney in the secondary fever of cholera. In the case of a woman who died in the sixth month of pregnancy we found the liquor amnii to be in the usual abundance, but cases, we believe, are related where it has been all absorbed.

The microbe found by Koch, and which he calls the "comma bacillus," is said to be characteristic. It has been found also by Klein and Gibbes in the dejecta and in the intestine, but they decline to say that it is the cause of the disease.

**Purpura.**—As a symptom, purpura may be found accompanying various general and local disorders, such as fevers and the exanthemata, as well as diseases of the liver, spleen, &c. In the absence of any such causes, we are obliged to look upon the affection as a specific one, and call it purpura. However, this is not common, but occasionally occurs, and sometimes constitutes a disorder which is fatal in a few days. As during life the whole body is covered with spots, and hæmorrhage occurs from every opening on the surface, so after death all the tissues of the body show effusions of blood, which is found to have burst forth from all parts of them. The serous and mucous surfaces are found covered with extravasated blood, also the bladder and kidneys, and in most cases also the brain; it may escape into the eye, causing blindness; the exudation of blood into the brain is often the immediate cause of death by interfering with important parts of the organ; in other cases death is caused by exudation of blood into the lungs, or simply by exhaustion; in some cases the spleen is found inordinately large, as well as its corpuscles.

In the last case we saw examined of a young woman the spleen was small, firm, and of a dark colour; at the same time there was no disease of any of the viscera. It must be remarked that in cases of acute purpura the temperature is often very high, which tends to corroborate the opinion that the cause of the disease must be found in some chemical change in the blood. In the case of a boy under Dr. Pye-Smith, which was fatal in two weeks with hæmorrhage into all parts, the swollen blood-vessels were found choked by micro-organisms or long chains of streptococci. A very similar condition was found in another case by Dr. Watson Cheyne. It was that of a young girl who had hæmorrhages into various parts of the body accompanied by a high temperature. In the spots of extravasation in the lungs and elsewhere he found the capillaries blocked with bacilli; the walls of the vessels were distended and ruptured. The bacilli seemed to act as emboli and plug the vessel until it burst. Purpura has a close relation to anæmia, for in some forms of the disease the patient may become anæmic and of a deadly pallor long before any hæmorrhage takes place.

**Hæmophilia.**—No cause has yet been found for this condition. The results may be seen in the hæmorrhages in the different parts of the body and in the joints. Dr. Theodore Acland reported the fatal case of a boy who was continually having hæmorrhages and in whom he found some peculiar changes in the thymus gland.

**Raynaud's Disease.**—This is a disease characterised by the appearance of black patches on the skin, which slough, leaving ulcers, and subsequently cicatrices, the prominent parts being more particularly affected, as ears, nose, and fingers. In a boy, lately in the hospital, the tip of the nose and ears turned black, the skin dried and shrivelled, leaving scars upon these organs. The tips of the fingers and toes at the same time turned black and shrivelled up. These eventually healed. He was also the subject of hæmatinuria.

**Scurvy.**—In this disorder the body is blotched with marks of hæmorrhage at different depths and of different ages, the superficial and recent patches being purple, while the older and deeper show the usual green and brown changes. We commonly find hard brawny tracts, generally in the limbs, especially about the knee. A section of these shows them to be due to effused blood, more or less altered; this blood may be in the muscles and intermuscular tissues, and rarely under the periosteum, sometimes in all these parts together. The joints may contain blood; the knee-joint may be distended with it; inflammation of the joint may have arisen as a consequence. Hæmorrhages, similar to those found in purpura, affect the great viscera and alimentary canal, whose interior may be blotched with ecchymoses, some of which may be found eroded. The heart is soft and pale, showing fatty degeneration within in many cases, and there may be embolic infarctions in the lungs or spleen; further, the lungs may show pneumonia; and the spleen is large and soft; the blood has a watery appearance.

**Anæmia.**—This disease, first described by Addison as "idiopathic anæmia," and subsequently by German writers as "progressive pernicious anæmia," is characterised by a pallor and bloodlessness, which gradually increases until death takes place, and as on microscopic examination no marked disease of any viscus is apparent, the affection is attributed to a change in the blood itself. This fluid resembles, to the naked eye, a pink-coloured water, and forms no coagulum in the heart or vessels. The microscope shows a great diminution of red corpuscles, even to the amount of one fourth, these also being altered in size and shape, some being larger than normal, and others putting on a pear or bottle shape. During life there is often œdema of the skin and a tendency to hæmorrhage, an effusion into the retina not being uncommon. After death there may be found serous exudation into the cavities as well as into the organs, rendering them œdematous. Ecchymoses may also be found in the various tissues, the brain being especially affected in this manner, although otherwise healthy. The heart presents the greatest morbid peculiarities, having on its inner surface, especially on the left ventricle, parallel white zigzag lines, or tabby mottling, as it is sometimes called; this is best seen on the columnæ corneæ. The heart is otherwise somewhat pale and flabby. The voluntary muscles throughout the body are often found slightly fatty. The stomach shows a degenerative change in the glands and an atrophy of the tubules. The liver is pale, and not infrequently has an excess of fat; the spleen shows no special change, and may be found of various sizes. It has been said that there is an increase of bone marrow in idiopathic anæmia, but this is questionable.

We have already mentioned under "Intestines" a fatal form of anæmia occurring in Egypt and some other countries, and which has been found due to a parasite—*Sclerostoma duodenale.*

**Leuchæmia.**—This is the case where an increase of white corpuscles exists in the blood, and is found pervading all the organs of the body.

How far there is an actual excess of white corpuscles or a diminution of the red has not been accurately ascertained. It is supposed that the blood-making organs are especially at fault, viz. the spleen and lymphatic glands; with them some would place the intestinal glands and the bone marrow; consequently an excessive quantity of white corpuscles are produced, which are deposited in other organs, as the kidney and liver. For further information see "Spleen."

**Myxœdema or Adult Cretinism.**—The first term is that given by Ord to designate the disease called sporadic cretinism by Gull, and cachexie pachydermique by some French writers. According to the first designation it is a progressive disease, in which the tissues of the body are invaded by a jelly-like or mucus-yielding material. The malady is most usually met with in women of middle age, who are observed to become lethargic; at the same time a remarkable change occurs in their appearance, the skin becoming swollen, pale, and translucent, the whole expression altered, the lips, nose, and ears becoming enlarged and rounded. Dr. Ord believes the essential character of the malady is the pouring out of a translucent albuminoid matter into the tissues of the skin and all the organs of the body, whilst others regard the change which takes place in the thyroid as the most essential part of the disease, for in most cases it is found either quite atrophied or so altered that none of the natural tissue remains. This association with disease of the thyroid is very remarkable, for when animals have undergone removal of this gland a condition resembling myxœdema has ensued; and also in the human subject, when the thyroid has been excised, the patients have fallen into a feeble, indolent condition, ending in fatuity. A case even has been recorded where such symptoms, or an acute myxœdema, came on very rapidly after removal of the thyroid body. Some French writers have been inclined to regard the disease as one rather dependent upon a lesion of the nervous system, especially the sympathetic. They have thought that the ganglionic nerves, by affecting the lymphatic vessels, might bring about the change. Seeing that the only constant change found in all cases which have hitherto been examined is disease of the thyroid body, many failing to discover the universal mucoid infiltration, the prevailing opinion at the present time is in favour of the disease we are describing being due primarily to atrophy or destruction of the thyroid, other morbid changes found being non-essential, such as thickening of the skin and more or less degeneration of the organs, as liver or kidney, and of the arteries.

**Sporadic Cretinism in Children, or Cretinoid Idiocy.**—This is an allied affection, and has been especially studied by Dr. Fagge. It has been observed that in all communities children sometimes cease to grow about the age of three or four, and soon come to resemble the cretins of the Alpine regions. In well-marked cases the child has an idiotic expression, the face being broad, nose flat, the mouth large, and the tongue often protruding; at the same time the limbs are short and curved, so that it has scarcely power to stand or walk. The thyroid body is absent, and on each side of the neck there exists a fatty tumour. Occasionally a child has been born presenting the appearance of a cretin, and one case has been reported where the condition was not observable until the boy was nine years old, when he grew fat and put on all the usual characters of this affection.

**Acromegalie** (*ακρον*, extremity).—This is a disease characterised by an overgrowth or gigantic enlargement of the hands and feet, together with a corresponding increase in the size of the features of the face. The name has been given by Dr. Marie, who observed several cases of the kind in the Salpêtrière. The disease had been noticed by others, but had been confounded with adult cretinism or osteitis deformans. Thus twenty years ago we had under our care a young lady whom we sent to Dr. Fagge, and who reported it in his work on medicine. Her features underwent a most extraordinary change: the mouth became enormous, like that of a negress, and the nose equally enlarged, the whole face being elongated. The hands became so large that no gloves could be found to fit her, and the feet also grew in the same proportion. She became blind and died at the end of six years. In some fatal cases reported from abroad there was found a growth in the pituitary body; there was also an absence of the thyroid gland.

**Exophthalmic Goitre.**—In the cases which have been examined at Guy's, as in those which have been reported from elsewhere, no marked organic disease has been found, all the organs appearing healthy. In some cases the heart may have been somewhat enlarged. The sympathetic in the neck has been most carefully examined, and has also been found healthy. The thyroid body has undergone hypertrophy and its blood-vessels have been correspondingly enlarged. Around these there has been found an increase of connective tissue.

**Diabetes.**—In diabetes the body is wasted, but hitherto no local change in explanation of the cause of the malady has been discovered. The cause of death is frequently found in the lungs. The brain has a healthy appearance. Dr. Dickinson has described minute degeneration following the course of the small vessels, and revealed by extravasations of blood, and some disintegration of the tissues, with the presence of granular corpuscles; all these he found in the medulla oblongata, cerebellum, pons, or more widely distributed. These appearances have been regarded by others as not characteristic but belonging to many wasting diseases. Since the discovery of artificial diabetes by wounding the floor of the fourth ventricle this part has been specially examined, but there are not more than three or four instances where obvious disease was found. Dr. Hale White has also examined the sympathetic nerves without any result. The liver, too, whose sugar-forming function (directly or indirectly) has drawn to it so much of the attention of physiologists, generally looks quite healthy; we have generally found it small, sometimes very small, certainly not looking as if it had been overworked; in section quite healthy, but devoid of fat. Diabetes, indeed, has seldom in our experience coexisted with severe disease of the liver, but in one case we found cirrhosis and in another cancer. On the other hand, the kidneys are large, indicative of the increased work they have had to perform, the pair often weighing sixteen to eighteen ounces; we have found the epithelium sometimes loaded with fat—good examples of simple fatty kidney, without any Bright's disease or other morbid condition.

The alimentary canal is generally healthy; the intestines usually filled with hard scybala; we have once, however, seen diabetes fatal by acute ileitis. The pancreas has frequently been found small and having undergone fatty degeneration; this has been noticed by ourselves and others, more especially by Lancereaux.

The lungs are nearly always diseased; sometimes death has taken place rapidly by pneumonia, which in one of our cases ended in gangrene; but generally the affection is an ordinary phthisis, of the pneumonic or tuberculo-pneumonic kind, vomicæ being present in the upper parts, and caseous pneumonia and tubercles in the lower parts. When death occurs suddenly, or from coma, there is no discoverable organic disease. Sometimes *lipœmia* exists, or a fatty state of the blood, shown by a white layer forming on its surface. Death has been supposed to be due in some cases to a collection of fat in the vessels or to "fat embolism." Several cases have been recorded where this was regarded as probable.

**Syphilis.**—We need not speak of primary syphilis, deaths from which are now very rare, but will consider the later affection of the great viscera, which indicates the general character and distribution of the disease, while it produces a cachexia that may develop a lardaceous state of the organs, and so be fatal by anæmia and albuminuria, or fastens on some vital organ, and by interfering with its structure destroys life. We have already, in connection with the separate viscera, spoken of changes and deposits which may be attributed to syphilis, and we will now class them together as a whole.

If one looks over a series of cases of syphilitic change, and compares these with any other forms of change, one finds that the syphilitic cases have characters by which they are practically easily distinguished. These characters we will attempt to state. Generally a small part of the organ is attacked, and the remainder is left quite free. The disease is strictly localised in the spot it affects. Its outer part is composed of fibrous tissue, which can be seen to represent the natural fibrous supporting elements of the part in a state of augmentation, while the functional elements of the part have dwindled away. It is a local sclerosis. Its central part shows the caseous or gummatous, faint yellowish, matter of more and more elastic consistence, and less and less friability and curdiness; generally rather sharply distinguished from the fibrous outer part, and sometimes softening down or calcifying. Sometimes there are signs of more acute inflammation in the immediate neighbourhood, showing lymph, &c., or adhesions to the parts around.

Such patches, sharply contrasting with more healthy tissue immediately about them, and distributed more or less widely in a variety of organs, but especially in the testes and liver, are not a general thing that could be passed over as a common accident. Their characters attract attention. A syphilitic gumma in muscle or brain is so unlike anything else that, if seen for the first time by one who knows the rest of the common run of pathological changes, it demands from him some recognition of its peculiarities. In short, it is not common, but specific in the strict sense of the word. We mean that we are not to suppose that these syphilitic changes are of a kind that are otherwise recognised as non-syphilitic, or in any sense of a "common" kind. It is not so. They are quite peculiar; the characters we have above enumerated do not belong to any other change, and amply suffice to distinguish practically the syphilitic from all other changes.

The general result of observations of the structural characters of syphilitic deposit in all parts of the body is the same, and to this effect, that there are few now who suppose that syphilitic formation presents any characteristic elements, or that the absence of characteristic elements invalidates the evidence of the syphilitic nature of these formations; these being ill-defined, although occasionally we may discover well formed cells.

But, on the other hand, syphilitic deposit often does not show these defined and well-characterised cells; it may even offer nothing more than common wavy areolar fibrous tissue, more or less deformed, with some fat-grains embedded, or some decayed nuclei present in it. These differences are partly a matter of stage or period of growth. The large and better-formed cells being new and young, the old fibrous and fatty material being at the period of full development, or senescence, or decay. Thus, a variety may be found in the liver where an isolated softened red patch constitutes the first stage; it is sometimes met with in cases where the next stages identify it. This red patch is, in the second stage, found to have a pale-yellowish subelastic tough substance forming in its middle, while around this substance, within the red patch, there is recent fibroid formation of the usual greyish cast of colour, the remains of the red soft patch encircling and spreading around these. Then, a third stage, a softening or a calcification of the yellowish centre, proceeds, or else a simple wasting away of it, which latter is more common; so that there usually remains at last a sunken scar in the place of the tissue, which has in this way been changed and removed. Such a series of states from the red patch to the calcified, softened, or merely scar-like relic, gives scope for great differences in the actual appearance of individual instances, and these stages in themselves would make syphilitic formations various enough. But all the differences in syphilitic deposits are not due to the stage or period of the growth.

In some cases the syphilitic formation wears very much the appearance of actively growing tumour. This is especially the case in the membranes and cortex of the brain. It forms a large and bold mass with rounded outline and fleshy consistence, enclosing in the middle some caseous change, but with a considerable proportion of it vascular and fleshy, so that from the appearance alone it could not be told whether the disease were a sarcous tumour or not; and one has to view the whole case and make a judgment, or at least arrive at a conviction, from the general characters of the other formations in the body, the history, &c.

We need not again detail the special appearances of syphilitic disease of the several organs and systems, as we have already described them when speaking of the organs themselves. When syphilitic formations are present, we often find lardaceous disease of the abdominal viscera. See cases detailed in 'Guy's Hospital Reports' for 1863 and 1867.

**Rickets.**—This is a condition of perverted nutrition, probably due to diet. Alterations are found in the cranium, thorax, and long bones, with decay of the teeth. These are described under the "Skeleton." Some pathologists believe that there are changes also in the liver, spleen, and lymphatic glands.

**Lardaceous Disease.**—We have described the lardaceous condition of the several organs especially liable to it; in all of them the change is essentially the same. It first affects the minute vessels, especially the arteries, and extends to the capillaries, afterwards invading the proper elements of the texture implicated. Its favourite seats are the kidney and spleen, which are about equally often lardaceous; the liver, which stands near them in liability; the lymphatic glands, which are frequently involved; the alimentary canal, which is not so generally obnoxious, yet often gives evidence of lardaceous disease, and is prominent on account of the obstinate symptoms which its disorder induces. After these parts we occasionally meet with it in the supra-renal bodies, the thyroid gland, and the tonsils. Following these, very far from them, are a number of other parts, such as the muscles, the membranes of the brain, the bladder, serous membranes, the nerves of the heart and of the uterus, which are said to be sometimes affected. Occasionally a new growth may undergo a lardaceous change.

As to the nature of the change, the peculiar reaction with iodine still remains almost the only distinctive or well-ascertained feature of the morbid process, although Drs. Ralfe and Pye-Smith have found the iodine reaction with other substances. In the 'Report of the Path. Soc. Committee,' 1872, Dr. Marcet says that iodine has the power of precipitating an aqueous infusion of a lardaceous liver, throwing down a dark matter, to which matter he ascribes the deep colour produced on lardaceous organs by the action of iodine. Dr. Marcet found that ultimate analysis of lardaceous parts showed less of nitrogen than is present in albumen, a fact which would rather argue that the amyloid substance is, after all, of the nature of ligneous or starchy matter. We must remember that any lardaceous tissue used for such analysis contains only a small relative part of the amyloid substance, inseparably engaged in the nitrogenous proper texture of the tissue. We have already alluded to Dr. Dickinson's hypothesis as to the nature of the amyloid material under "Liver," his theory leading us to believe that some new material in the blood is poured out into the tissues, as, for example, into the liver, and that this may be again absorbed, leaving the organ healthy. Most pathologists, however, regard the condition as a degeneration rather than an infiltration, seeing that some tissues are more affected than others, as if they had some affinity for certain elements in the blood with which they might combine; therefore the lardaceous material is formed in the organ itself.

We have never seen any facts to show that lardaceous change leads to a fibrous scar-like withering, as some have described; and we believe the scar-like patches in question to have been due to syphilis, a frequent concomitant and cause of lardaceous action.

Amongst the causes of lardaceous disease syphilitic and scrofulous disorders of the bones are most prominent. Chronic suppurative bone disease is so frequently the cause that one naturally examines the bones to ascertain if the matter has arisen there, but the bones do not then yield the amyloid reaction. It has been said, however, that the lymphatic glands corresponding to the diseased bone are first implicated; this we are not able to confirm; we have several times found the corresponding glands free when the viscera were much involved in the disease. Late syphilis, without any suppuration, will certainly produce profound amyloid change. So, it is said, will malarious fever, but there is, we think, suspicion that syphilis complicated the cases published by Frerichs in proof of this; the description of the liver strongly infers syphilitic disease in those cases. Otherwise the amyloid affection has almost always attended upon prolonged suppuration, such as that in phthisis, in scrofulous disease of the kidneys, in old dysenteric ulceration, in old obstinate carbuncle, leprosy, &c.

**Burns.**—Persons not destroyed by the heat and suffocation, often, if the burn is severe, die in the course of twenty-four to forty-eight hours. The destruction of a considerable part of the skin will then be seen, but usually the viscera are found to all appearance healthy; or there may be in rarer cases evidence of bronchitis. In cases that survive several days we may find broncho-pneumonia, or catarrhal nephritis, or deep ulceration of the duodenum, perhaps opening large arteries or perforating the peritoneum ; more rarely some other part of the alimentary canal may be ulcerated. Yet later the usual morbid anatomy of pyæmia may be found.

**Suffocation, Strangulation, Drowning.**—In death from any of these causes the surface of the body shows marked lividity, the great viscera are found loaded with dark blood, and there may be some effusion of blood on the brain, or in the air-passages and lungs, or under the pleura. The blood is everywhere liquid, as usual in sudden death. The heart is generally contracted firmly, so that its left ventricle is empty, while the right cavities, which close less completely and are thinner, contain a larger quantity of fluid blood ; there is much of this fluid blood in the large systemic veins. This observation should be made before removing the lungs from the body, but not before we have become familiar with the amount usually present in ordinary cases. Froth may be present in the trachea in death from any of these causes ; this froth may be tinged with blood. Thus, the results of internal examination in such cases, which are often separately enumerated as though distinctive, will, so far, not enable us to distinguish between these forms of death, nor, indeed, to distinguish between them and death from epilepsy, &c. Whence it is the more necessary that we should watch keenly those incidental conditions of and about the body which may enable us to come to a conclusion as to the cause of death.

*Death by suffocation.*—The suspicion of death by suffocation should induce us to thoroughly examine the whole tract of the breathing passages ; the nasal cavities being carefully inspected for foreign bodies in some form of dust, which may have been drawn in, and, if found, may indicate the circumstances of the accident. The tongue, too, which often protrudes during suffocation, should be looked to ; its tip may be found covered with dust, &c., although now it may be withdrawn into the mouth. The fauces should, of course, be carefully examined. We have met with a case where a man was brought in dead from a public-house, and there was no other disease than a monstrous lump of half-chewed steak, quite filling the upper part of the pharynx. In cases of cerebral disease, especially in softening of the pons, such choking may be found. In suffocation by poisonous sewer-gases the pupils have been found dilated. When the bodies are found some hours after death, and have not received the customary nursing attentions, the eyes are, of course, usually found open.

*Strangulation.*—In death by strangulation the mark of the cord or other means of compression forms the only ground of diagnosis afforded by the body. The marks correspond pretty closely with the points of rough contact of the fatal means. In hanging they are found chiefly behind the ear, about the occiput ; in strangling with cords they encircle the neck; pressure with the fingers leaves the imprint of these. Such marks are not absolutely always, but are pretty generally present. The marks themselves are seen in a dirty yellowish dryness of the spot, which is hardish, and has partly lost its epidermis. This is the case if the body has lain many hours, but at an earlier period, or in slighter degrees of injury, the mark may be livid, with folds, creases, &c., printed by the ligature; blood may possibly be effused beneath it. Experiment has shown that all these effects may be produced by suspension or ligature, on bodies recently dead. In some cases of *hanging*, great damage is done to the deep textures, the neck being dislocated or broken and the soft parts torn; but death more frequently, in suicidal cases, results from asphyxia.

*Drowning.*—In the examination of drowned persons it is very important to note minutely the state of the surface of the body, observing any scraps of weeds, mud, &c., that may be found under the nails or clinging to the surface; also to inspect very carefully any apparent bruises or other injuries. All this, with the state of the surface of the body, showing the effects of continuous soaking in water, and the laws that determine decomposition, according to the period of the year, and the running or stagnant condition of the water, will belong more naturally to the compass of works on jurisprudence.

In the internal examination we look for the presence of water in the air-tubes. It has been disputed whether the water in which the person is drowned finds entry, but this undoubtedly occurs. Also in the stomach and in the œsophagus, such water, having in it, perhaps, weeds, &c., may be found. In shifting and carrying the body the contents of the stomach may find their way into the windpipe; this can generally be known by their strongly acid reaction. It appears from some carefully recorded cases that death may take place through shock, when persons are thrown or fall into the water, so that the characters of asphyxia are not produced. The same remark applies to deaths by suffocation and strangulation, for in these we do not always meet with the lividity and general congestion which you would anticipate. We believe this to be more common than is generally supposed. If pressure be made on the neck of a person standing upright he will immediately fall, and if the experiment be made on an animal of placing ligatures on the blood-vessels in the neck there is immediate loss of consciousness. If death therefore takes place from strangulation there may be none of the usual appearances of asphyxia present. Persons saved from drowning after falls in the water often die in a few days from acute pneumonia, as we might expect from the congestion of the lung, often so great that hæmoptysis follows, especially remembering the water breathed into it and the chill and the shock which the person has undergone.

**Lightning Shock.**—In sudden death from lightning no abnormal appearance is necessarily found, and that no serious lesion need occur is seen by such a case as we had once in the hospital, when a woman was brought in unconscious and collapsed, but was quite well again in three days. It has been said that no rigor mortis is present, and that the blood is always coagulated, but in one case which we had an opportunity of seeing rigor mortis existed several hours after death, and the blood was everywhere fluid. Very often, however, there is some positive lesion, as in the case of a man who was struck down in the Borough, and picked up hemiplegic. In his case it was found that a severe laceration of one side of the brain had occurred. A very remarkable case was brought to the Clinical Society by Mr. Wilks, of Ashford. A man, standing under a tree, was struck by lightning; the latter had its bark stripped off, while the man was found lying two yards off almost naked, crying for help. His clothes were torn into shreds and strewn about the field. His trousers were hanging in pieces upon him, his boots had been dragged off his feet, and his watch and chain were fused. He was burnt and lacerated all over, and one leg was broken. He had never lost his consciousness and slowly recovered. In other cases the skin has been burned, sloughing and ulceration has followed, leaving deep cicatrices.

**Sunstroke.**—Sometimes persons exposed to the sun fall unconscious, and death rapidly occurs from syncope. In other cases death is not sudden, a rise of temperature taking place, the patient subsequently succumbing from hyperpyrexia. A post-mortem examination has shown nothing more than a gorged venous system, as if death was due to asphyxia, there being no cerebral hæmorrhage whatever. The brain, however, is evidently often damaged in *coup de soleil*, as seen by the various cerebral symptoms which may follow slighter attacks of sunstroke, such as fits, giddiness, &c.

**Poisons.**—The effects of poisons, so far as they are anatomically evident, we have described under the several viscera affected by the poison.

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